Snippet

In the absence of insulin , heat_shock caused a significant increase of alpha_2M and AOG secretion .

Angiotensin-converting_enzyme (ACE) activity has been determined in the semen of certain avian and mammalian species as well as its release during cold_shock .

The study provides the first evidence of a possible direct effect of heat_shock on adiponectin and leptin gene expression and secretion , and demonstrates that the expression of the two adipokines is differentially regulated at the temperatures tested .

In this study , we investigated the effect of heat_shock on gene expression and secretion of adiponectin and leptin , and gene expression of Hspa2 and Ppargamma in 3T3-L1 adipocytes .

RESULTS : In SP , adiponectin was significantly lowered and resistin , active PAI-1 , MCP-1 , IL-1_alpha , IL-6 , IL-8 , IL-10 , and TNF-alpha were significantly elevated compared to BD .

In SP , adiponectin correlated negatively with BMI , SAPS II and SOFA scores , while resistin correlated positively with SAPS II and SOFA scores and leptin correlated positively with the BMI .

OBJECTIVE : To investigate plasma concentrations of adrenomedullin in patients with septic_shock and the potential association of these concentrations with relaxation of vascular tone .

Increased plasma concentrations of adrenomedullin correlate with relaxation of vascular tone in patients with septic_shock .

Increased adrenomedullin concentration in cerebrospinal fluid in patients with septic_shock .

In univariable analysis , elevated levels of soluble RAGE on admission were associated with adverse outcome , including circulatory_failure , kidney_failure , liver_dysfunction , and mortality .

High-mobility_group_box_1 (HMGB1) and receptor for advanced glycation end-products (RAGE) axis is also involved in septic_shock .

Induction of a heat_shock response and down-regulation of Akt and Raf-1 were observed in biomarker studies .

Following heat_shock , the Akt level decreased by 15-70 % in a temperature-dependent and phosphorylation status-independent manner .

Albumin has always been used in many clinical states especially to improve circulatory_failure .

In the subgroup of 85 patients with nosocomial BSI , the albumin : PCT ratio in patients with shock was lower than that in those without shock (7.154 [2.975-26.267] vs 28.000 [3.818-57.812] ; p = 0.027) .

Compared with alternative treatments , albumin reduced the incidence of postparacentesis circulatory_dysfunction (odds ratio [OR] , 0.39 ; 95 % confidence interval [CI] , 0.27-0.55) .

[Use of serum_albumin in the complex treatment of traumatic_shock during first aid] .

In contrast , Ang-1 decreased up to 35-fold in CS (P < 0.001) .

We hypothesized that plasma angiopoietin-1 and angiopoietin-2 levels are associated with indirect markers of increased vascular permeability , organ dysfunction , mortality , and plasma proinflammatory cytokine levels in human septic_shock .

A high angiopoietin-2 / angiopoietin-1 ratio is associated with a high risk of septic_shock in patients with febrile_neutropenia .

Angiopoietin-2 emerged as an independent predictor of 28-day and 1-year mortality in CS .

[Angiopoietin-2 : prognostic parameter in cardiogenic_shock] .

CONCLUSION : In CS , high levels of Ang-2 are independently associated with poor short - and long-term outcome and associated with the reperfusion success as well as complications .

[Angiopoietin-2 as a mediator of capillary leaks in septic_shock : `` mind the gap ''] .

CONCLUSIONS : Increased plasma angiopoietin-2 levels are associated with increased fluid overload , hepatic_and_coagulation_dysfunction , acute_kidney_injury , mortality , and plasma cytokines in human septic_shock .

Plasma levels of endothelial biomarkers as angiopoietin-2 , sE-selectin or endocan were measured at ICU admission of 20 patients presenting with septic_shock .
We have previously reported elevated levels of angiopoietin-2 in patients with septic_shock , and have investigated tumor_necrosis_factor (TNF) - related and weak inducer of apoptosis (TWEAK) , which mediates both angiogenesis and inflammation , in those patients .
CONCLUSIONS : In contrast to VWF , plasma angiopoietin-2 positively relates to fluid balance , pulmonary_dysfunction and mortality throughout the course of septic_shock , in line with a suggested mediator role of the protein .
PURPOSE : To investigate whether angiopoietin-2 , von_Willebrand factor (VWF) and angiopoietin-1 relate to surrogate indicators of vascular permeability , pulmonary_dysfunction and intensive care unit (ICU) mortality throughout the course of septic_shock .
Circulating angiopoietin-2 levels in the course of septic_shock : relation with fluid balance , pulmonary_dysfunction and mortality .
The median Ang-2 was higher in septic_shock .
The authors found that an increased Ang-2 / Ang-1 ratio , or an elevated Ang-2 level , at the time of an initial fever , is associated with subsequent development of septic_shock and death .
In contrast , Ang-2 concentrations were increased in patients with septic_shock , whereas an inverse finding was observed for Ang-1 , resulting in a higher Ang-2 / Ang-1 ratio in patients with septic_shock (5.29 , range 0.58 to 57.14) compared to non-complicated FN (1.99 , range 0.06 to 64.62 ; P = 0.01) .
Ang-2 was higher in patients with septic_shock compared to patients with sepsis , severe sepsis and controls .
CONCLUSIONS : APC levels are increased in patients with septic_shock and are correlated with established markers of coagulation .
The evolution of activated_protein_C plasma levels in septic_shock and its association with mortality : A prospective observational study .
OBJECTIVE : To investigate the effects of activated_protein_C (APC) in a clinically relevant animal model of septic_shock .
Beneficial effects of recombinant human activated_protein_C in a ewe model of septic_shock .
Serum Apelin level was significantly higher in patients with septic_shock than those with severe sepsis ($P < 0.01$) .
In conclusion , we demonstrated that heat_shock decreased AQP5 on cellular membranes and in the cytoplasm by activating autophagic degradation , and heat_shock and AQP5 knockdown exerted similar anticancer effects , suggesting that heat_shock exerts anticancer effects via the autophagic degradation of AQP5 .
During vasodilatory_shock after prolonged and severe hemorrhage , VP seems to be effective in reversing hypotension and decreasing the need for exogenous cathecholamines while preserving cardiac function and critical organ blood flow .
VP was infused in two posthemorrhagic vasodilatory_shock patients when they remained persistently hypotensive despite adequate fluid resuscitation and infusions of pharmacological doses of catecholamines .
CONCLUSIONS : Vasopressin is an effective pressor in vasodilatory_shock after cardiopulmonary bypass .
CONCLUSIONS : Vasopressin plasma levels are inappropriately low in vasodilatory_shock , most likely because of impaired baroreflex-mediated secretion .
CONCLUSION : Vasopressin (0.01-0.04 U/min , IV) should be considered in small animal veterinary patients with vasodilatory_shock that is unresponsive to fluid resuscitation and catecholamine (dobutamine , dopamine , and norepinephrine) administration .

Vasopressin decreases intestinal mucosal perfusion : a clinical study on cardiac surgery patients in vasodilatory_shock .
Vasopressin effective in reversing catecholamine-resistant vasodilatory_shock .
CONCLUSIONS : Vasopressin infusion improved the hemodynamic state in advanced shock without compromising cardiac function .
SUMMARY : Vasopressin administration is very effective in restoring arterial pressure in many forms of shock and this appears to be due , at least in part , to deficiency of endogenous hormone .
Vasopressin (antidiuretic_hormone) is emerging as a potentially major advance in the treatment of a variety of shock states .
Vasopressin vs Terlipressin in Treatment of Refractory_Shock .
Vasopressin and corticosteroids may have a role in reversing refractory_shock and work primary through nonadrenergic mechanisms .
Vasopressin was introduced as a final attempt to reverse the refractory_shock and was associated with recovery .
Safe Use of Vasopressin and Angiotensin_II for Patients with Circulatory_Shock .
To clarify the hemodynamic effects of vasopressin on pediatric patients with vasodilatory_shock after cardiopulmonary bypass , 70 consecutive patients , most of whom with right_heart_anomalies , were retrospectively analyzed in Fuwai Hospital from October 2013 to September 2015 .
Although the use of vasopressin has become commonplace in pediatric patients with vasodilatory_shock after cardiac surgery , its efficacy and hemodynamic effects have not been systematically documented .
In conclusion , low dose of vasopressin administration was associated with great and timely hemodynamic improvement for pediatric patients with vasodilatory_shock after cardiac surgery without any significant adverse effects .
OBJECTIVE : To discuss 3 potential mechanisms for loss of peripheral vasomotor tone during vasodilatory_shock ; review vasopressin physiology ; review the available animal experimental and human clinical studies of vasopressin in vasodilatory_shock and cardiopulmonary_arrest ; and make recommendations based on review of the data for the use of vasopressin in vasodilatory_shock and cardiopulmonary_arrest .
The use of vasopressin for treating vasodilatory_shock and cardiopulmonary_arrest .
BACKGROUND : Low to moderate doses of vasopressin have been used in the treatment of cathecholamine-dependent vasodilatory_shock in sepsis or after cardiac surgery .
The vasopressin and copeptin response in patients with vasodilatory_shock after cardiac surgery : a prospective , controlled study .
The purposes of this review are to outline the pathophysiology of vasodilatory_shock after cardiopulmonary bypass , to discuss the physiological role of endogenous vasopressin , to explore the clinical basis for vasopressin replacement , and to review the pharmacology and dosing guidelines .
The experience with this case suggests that vasopressin may be a valuable adjunct to the treatment of catecholamine-resistant vasodilatory_shock .
The high incidence of vasodilatory_shock in patients undergoing left_ventricular_assist_device (LVAD) implantation makes this population an ideal model in which to assess the risks and benefits of vasopressin .
After 10 years of ongoing research , vasopressin has grown to a potential component as a vasopressor agent of the anesthesiologist 's armamentarium in the treatment of cardiac_arrest and severe shock states .
PATIENTS AND PARTICIPANTS : Patients (n = 117 ; 32 noncardiac , 85 postcardiac surgery) requiring intravenous vasopressin infusion longer than 60 min for advanced shock (January 2004 to December 2005) .

OBJECTIVE : To study effects of vasopressin on hemodynamic , clinical , and laboratory variables in children with advanced vasodilatory shock .
RECENT FINDINGS : Examples of types of shock resistant to catecholamine pressors in which exogenous vasopressin was effective in restoring arterial pressure continued to accumulate .
The present review focuses on recent work that addresses the role of endogenous vasopressin in the pathogenesis of shock and the potential therapeutic indications and secondary effects of exogenous hormone in patients with shock .
This article reviews the physiology and pharmacology of vasopressin and all of the relevant animal and human clinical literature on its use in the treatment of shock following a MEDLINE (1966-2000) search .
There is current interest in the use of vasopressin in the treatment of shock due to ventricular_fibrillation , hypovolaemia , sepsis and cardiopulmonary bypass .
Although vasopressin has similar direct actions to the catecholamines , it may uniquely also inhibit some of the pathologic vasodilator processes that occur in shock states .
From animal models of shock , vasopressin has been shown to produce greater blood flow diversion from non-vital to vital organ beds (particularly the brain) than does adrenaline .
Increasing interest in the clinical use of vasopressin has resulted from the recognition of its importance in the endogenous response to shock and from advances in understanding of its mechanism of action .
While vasopressin has in the past been primarily used in the management of diabetes_insipidus and acute_gastrointestinal_bleeding , an increased understanding of the physiology of refractory_shock , and the role of vasopressin in maintaining cardiovascular homeostasis prompted a renewed interest in the therapeutic roles for this hormone in the critical care setting .
Role of vasopressin and terlipressin in refractory_shock compared to conventional therapy in the neonatal and pediatric population : a systematic review , meta-analysis , and trial sequential analysis .
Arginine vasopressin is frequently used in refractory_shock , although definite evidence to support this practice is still missing .
Nevertheless , available clinical data supports the use of vasopressin in critically ill children as a rescue therapy in refractory_shock and cardiac_arrest .
Small bowel and liver tissue pO2 and pCO2 during hypovolaemic_shock and intravenous vasopressin infusion .
OBJECTIVE : To assess the sublingual microcirculation in a patient during vasopressin administration for a distributive_shock after cardiopulmonary bypass .
Effect of vasopressin on sublingual microcirculation in a patient with distributive_shock .
CONCLUSION : Plasma copeptin levels are elevated in patients with advanced vasodilatory_shock .
In SIRS patients without shock , serum osmolarity was indirectly associated with AVP levels , whereas mean arterial blood pressure and serum osmolarity were associated with AVP in SIRS patients with shock .
In SIRS patients without shock , serum osmolarity was indirectly associated with AVP levels , whereas mean arterial blood pressure and serum osmolarity were associated with AVP in SIRS patients with shock .
CONCLUSION : Post-cardiopulmonary bypass vasodilatory_shock is largely due to a relative deficiency of Arginine_vasopressin .
RESULTS : A relative or absolute deficiency of Arginine_vasopressin is associated with vasodilatory_shock after cardiopulmonary bypass .
Antidiuretic_hormone replacement therapy to prevent or ameliorate vasodilatory_shock .
Vasopressin infusion of 0.01 to 0.04 U/min in patients with septic_shock increases plasma vasopressin levels to those observed in patients with hypotension from other causes , such as cardiogenic_shock .

One patient experienced cardiogenic_shock from vasopressin , two developed postoperative_infections , and one was found to have adenomatous_adenomyosis instead of a leiomyoma .
CONCLUSIONS : In adults with septic_shock treated with concomitant VP and NE therapy , discontinuing VP first may lead to a higher incidence of hypotension but is not associated with mortality or ICU LOS .
METHODS : We conducted a retrospective analysis comparing three group of septic_shock patients based on the intervals of actual body mass index (BMI) in patients enrolled in the VASST (Vasopressin_and_Septic_Shock_Trial) cohort .
This review of vasopressin in septic_shock differs from previous reviews by providing more information on the physiology and pathophysiology of vasopressin and vasopressin receptors , particularly because of recent interest in more specific AVPR1a agonists and new information from the Vasopressin_and_Septic_Shock_Trial (VASST) , a randomized trial of vasopressin versus norepinephrine in septic_shock .
This review of vasopressin in septic_shock differs from previous reviews by providing more information on the physiology and pathophysiology of vasopressin and vasopressin receptors , particularly because of recent interest in more specific AVPR1a agonists and new information from the Vasopressin_and_Septic_Shock_Trial (VASST) , a randomized trial of vasopressin versus norepinephrine in septic_shock .
Hemodynamic Instability Secondary to Vasopressin Withdrawal in Septic_Shock .
Hemodynamic Instability Secondary to Vasopressin Withdrawal in Septic_Shock .
The Effect of Vasopressin on Gastric Perfusion in Catecholamine-Dependent Patients in Septic_Shock .
RATIONALE : Vasopressin may be used to treat vasodilatory_hypotension in septic_shock , but it is not recommended by guidelines as a first or second-line agent .
Bench-to-bedside review : Vasopressin in the management of septic_shock .
BACKGROUND : Vasopressin is commonly used as an adjunct to catecholamines to support blood pressure in refractory septic_shock , but its effect on mortality is unknown .
Vasopressin infusion in children with catecholamine-resistant septic_shock .
Vasopressin but not norepinephrine improved renal blood flow and oxygen delivery and prolonged survival in animal models of septic_shock .
Our first hypothesis was that the vasopressin versus norepinephrine comparison and 28-day mortality of patients with Septic_Shock_3.0_definition (lactate > 2 mmol/L) differ from vasopressin versus norepinephrine and mortality in Vasopressin and Septic Shock Trial .
PURPOSE : We performed an individual patient data meta-analysis to investigate the possible benefits and harms of vasopressin therapy in adults with septic_shock both overall and in pre-defined subgroups .
CONCLUSIONS : Discontinuation of norepinephrine prior to vasopressin during the recovery phase of septic_shock resulted in less clinically significant hypotension but no difference in mortality or lengths of stay .
Use of vasopressin in the treatment of refractory septic_shock .
METHODS : Single-center , retrospective cohort of patients receiving fixed-dose vasopressin for septic_shock for at least 6 h with concomitant catecholamines in the medical , surgical , or neurosciences intensive care unit (ICU) at a tertiary care center .
WHAT IS KNOWN AND OBJECTIVE : Despite widespread use of vasopressin for the treatment of septic_shock , few cases of diabetes_insipidus (DI) following its discontinuation have been reported . This study evaluates the optimal sequence for the discontinuation of vasopressin therapy in septic_shock .

The median hospital rate of vasopressin use during septic_shock was 11.7 % (range 0 to 69.7 %) .
Although vasopressin (VP) is recommended for the treatment of NA-resistant septic_shock , the optimal parameters for its administration remain unclear .
The objective of this study was to evaluate the effect of vasopressin on lactate and lactate clearance as markers of tissue perfusion during septic_shock .
CONCLUSIONS : Early initiation of vasopressin therapy in adult critically ill patients with septic_shock was associated with no difference in total catecholamine requirements but decreased incidence of new onset arrhythmias .
The vasopressin in septic_shock trial (VASST) compared the addition of vasopressin to norepinephrine alone in patients with septic_shock .
Further clinical , pharmacokinetic and pharmacodynamic studies are needed to better define the role of vasopressin and terlipressin in septic_shock , as well as to prove their effectiveness and safety in infants and children .
Predictors of prolonged vasopressin infusion for the treatment of septic_shock .
The interaction of vasopressin infusion and corticosteroid treatment in septic_shock requires further study .
However , vasopressin levels are inappropriately low in septic_shock .
Many small clinical studies of vasopressin infusion in septic_shock have shown that vasopressin infusion increases blood pressure , decreases requirements for norepinephrine and improves renal function .
INTRODUCTION : This study compares recent vasopressin use and outcomes to our early practice when vasopressin was introduced for septic_shock .
Patients recently treated with vasopressin have a higher SICU survival rate than the survival rate when vasopressin was first introduced for septic_shock .
OBJECTIVE : To compare the effects of vasopressin versus norepinephrine infusion on the outcome of kidney_injury in septic_shock .
The effects of vasopressin on acute kidney_injury in septic_shock .
Continuous terlipressin versus vasopressin infusion in septic_shock (TERLIVAP) : a randomized , controlled pilot study .
Low-dose vasopressin did not reduce mortality more than norepinephrine in septic_shock .
Effects of vasopressin in septic_shock .
Several clinical studies in adults and children have reported that the effects of vasopressin for the treatment of vasodilatory septic_shock , due to a variety of causes , are both beneficial and safe .
Ten years of vasopressin research in septic_shock : constant dripping wears the stone .
Terlipressin : vasopressin analog and novel drug for septic_shock .
Are vasopressin levels increased or decreased in septic_shock ?
We close by suggesting that further randomized controlled trials of vasopressin in septic_shock are required before vasopressin is used routinely for management of septic_shock .
We then highlight the areas of uncertainty in using vasopressin for septic_shock and summarize the reasons for clinical equipoise .
SUMMARY : There is growing evidence that vasopressin infusion in septic_shock is safe and effective .
Clinical trials are underway to determine whether administration of vasopressin can improve outcomes in patients with septic_shock .
The role of vasopressin in vasodilatory septic_shock .
Comment on `` Role of vasopressin in the management of septic_shock '' by Mutlu and Factor .
BACKGROUND : Small studies have reported that vasopressin improves hemodynamic_instability in patients with septic_shock .

The use of vasopressin for the treatment of septic_shock is increasing .
A long-acting vasopressin analog for septic_shock : brilliant idea or dangerous folly ?
The effect of vasopressin on gastric perfusion in catecholamine-dependent patients in septic_shock .
CONCLUSIONS : Plasma vasopressin levels are almost always increased at the initial phase of septic_shock and decrease afterward .
In septic_shock , vasopressin levels are low , and therefore , vasopressin has been advocated as a vasopressor .
CONCLUSION : These data suggest that in septic_shock , inappropriately low plasma levels of vasopressin are at least partly related to a depletion of vasopressin stores in the neurohypophysis .
OBJECTIVES : To assess the mechanisms underlying the inappropriately low plasma vasopressin levels reported in septic_shock .
OBJECTIVE : To review all cases of septic_shock treated with vasopressin to determine the effects on hemodynamic and renal function and to document any adverse effects .
PURPOSE : To evaluate the association between concomitant arginine-vasopressin (AVP) / hydrocortisone therapy and mortality in severe septic_shock patients .
OBJECTIVE : To compare the effects of arginine-vasopressin (AVP) and norepinephrine (NE) on hemodynamic variables , organ dysfunction , and adverse events in early hyperdynamic_septic_shock .
Moreover , HBP levels in patients with septic_shock were significantly higher than in patients with sepsis without shock (median 153.8 ng/mL vs 49.7 ng/mL , $p < 0.01$) .
CONCLUSIONS : Albumin inhibits heparin-binding_protein-induced increased human endothelial cell permeability and heparin-binding_protein greater than 30 ng/mL and heparin-binding_protein-to-albumin ratio greater than 3.01-but not serum albumin-identified patients at increased risk for acute_kidney_injury in septic_shock .
Levels of HBP were higher in patients with non-septic_shock and septic_shock than healthy controls .
Value of Serum Cholinesterase Activity in the Diagnosis of Septic_Shock Due to Bacterial_Infections .
BACKGROUND : We aimed to investigate whether serum cholinesterase (SChE) activity can be helpful for the diagnosis of septic_shock and to evaluate its usefulness in comparison with procalcitonin (PCT) and C-reactive_protein (CRP) .
[Cholinesterase in surgery (a comprehensive study on the behavior of the enzyme after operations and on the usefulness of enzyme control for the prognosis , prophylaxis and therapy of surgical_shock] .
[Cholinesterase activity in death due to traumatic_shock , craniocerebral_injury and stress] .
[Brain cholinesterase activity in traumatic_shock] .
[Therapeutic use of exogenous cholinesterase in traumatic_shock] .
The expression of BCL-2 interacting cell death suppressor (BIS) , an anti-stress or anti-apoptotic protein , has been shown to be regulated at the transcriptional level by heat_shock factor 1 (HSF1) upon various stresses .
However , pharmacological inhibition of BCL-2 and BCL-X (L) with ABT-737 also sensitized cells to heat_shock , most likely through liberation of BIM .
Stress-induced in vitro apoptosis of native human acute_myelogenous_leukemia (AML) cells shows a wide variation between patients and is associated with low BCL-2 : Bax ratio and low levels of heat_shock protein 70 and 90 .
Protein levels of B-cell_lymphoma_protein-2 (BCL-2) , BCL-x (L/S) , heat_shock protein 70/90 , and BCL-2-associated death protein remained unaltered .
We found that mild heat_shock upregulates not only HSP70 but also BCL-2 , though BCL-2 has not previously been recognized as a heat-inducible protein .

Heat-shock_proteins attenuate SERCA inactivation by the anti-apoptotic protein Bcl-2 : possible implications for the ER Ca2 + - mediated apoptosis .
Furthermore , we report evidence that impaired expression of CK2 results in destabilization of the Bcl-2 mammalian homolog Bcl-xL , which is known to stabilize the mitochondrial membrane potential , through a mechanism involving disruption of the chaperone function of heat_shock protein 90 .
The key resistance targets that are discussed include (1) Bcl-2 and Mcl-1 proteins ; (2) autophagy processes ; (3) necrosis and necroptosis ; (4) heat_shock protein signaling ; (5) the proteasome pathway ; (6) epigenetic mechanisms ; and (7) aberrant nuclear export signaling .
[Apoptosis-modulating effects of heat_shock proteins : the influence of Hsp27 chaperone on TBA Bcl-2 family proteins in Jurkat cell line] .
In vitro studies (T84 , HT29 , NCM460 human colon cell lines) examined colostrum effects on temperature-induced apoptosis (active caspase-3_and_9 , Baxa , Bcl-2) , heat_shock protein 70 (HSP70) expression and epithelial electrical resistance .
Bcl-2-associated_athanogene (BAG) family proteins are characterized by their property of interaction with a variety of partners involved in modulating the proliferation/death balance , including heat_shock proteins (HSP) , Bcl-2 , Raf-1 .
Bcl-2-associated_athanogene (BAG) family proteins are characterized by their property of interaction with a variety of partners involved in modulating the proliferation/death balance , including heat_shock proteins (HSP) , Bcl-2 , Raf-1 .
Bax , Bcl-2 , caspase-3 , caspase-9 , heat_shock protein (Hsp) 90 , p53 , p-p53 , p21 , Poly _ (ADP-ribose) _ polymerase (PARP) , and the inhibitor of caspase-activated_DNase (ICAD) protein expressions were detected by Western blot analysis .
Moreover , we found by mass spectrometry and co-immunoprecipitation assay that heat_shock protein 90b (Hsp90b) forms a complex with Bcl-2 in mast cells .
Moreover , antiapoptotic mechanisms (e.g. , Bcl-2 / Bcl-x proteins , heat_shock proteins) are equally effective in protection against apoptosis and necrosis .
The antiapoptotic mechanism can be understood via expression of protective genes such as heat_shock proteins , Bcl-2 as well as direct inhibition of the apoptotic caspase family proteases by S-nitrosylation of the cysteine_thiol .
The proteasome mediated step (s) in apoptosis is located upstream of mitochondrial changes and caspase activation , and can involve in different systems Bcl-2 , Jun_N-terminal_kinase , heat_shock proteins , Myc , p53 , polyamines and other factors .
Overexpression of bcl-2 suppressed glucose_deprivation-induced HSP70 gene expression , heat_shock transcription factor-heat_shock element binding activity , as well as c-Jun NH2-terminal kinase (JNK1) activation .
Regulation by bcl-2 , c-myc , and p53 of susceptibility to induction of apoptosis by heat_shock and cancer chemotherapy compounds in differentiation-competent_and _ - defective_myeloid_leukemic cells .
bcl-2 expression does not alter either the steady-state or heat-induced expression of heat_shock proteins in cells , nor is bcl-2 itself a stress-inducible protein .
The mechanism of stress resistance conferred by the bcl-2 alpha protein is yet to be determined although the resistance does not seem to be the result of an increase in major heat_shock proteins , hsp70 and hsp90 , nor the arrest of cells in G1/G0 phase .
This revealed that high levels of bcl-2 alpha protein made EBV-B cells more resistant to a variety of stresses including the application of heat_shock , ethanol , methotrexate and the absence of serum .
Fatty_acid synthase , bcl-2 , bcl-x , p53 , estrogen and progesterone receptors , heat_shock protein 60 and Her2-neu (c-erbB-2) were evaluated in a group of 149 breast_carcinoma patients with a 5-year follow-up period .

Actually , many investigators have been studied treatment strategies of cerebral_infarction using a variety of neurotrophic factors such as bcl-2 , heat_shock protein 72 , glial_cell_line-derived_neurotrophic_factor (GDNF) , and hepatocyte_growth_factor (HGF) .
Evaluation of HER2 , p53 , bcl-2 , topoisomerase II-alpha , heat_shock proteins 27 and 70 in primary breast_cancer and metastatic ipsilateral axillary lymph nodes .
Protein levels of B-cell_lymphoma_protein-2 (BCL-2) , BCL-x (L/S) , heat_shock protein 70/90 , and BCL-2-associated death protein remained unaltered .
Genetic variants in SERPINA4 and SERPINA5 , but not BCL2 and SIK3 are associated with acute_kidney_injury in critically ill patients with septic_shock .
BCL2 genetic variants are associated with acute kidney_injury in septic_shock * .
Protein levels of B-cell_lymphoma_protein-2 (BCL-2) , BCL-x (L/S) , heat_shock protein 70/90 , and BCL-2-associated death protein remained unaltered .
Thus , BPI is a promising therapy in the treatment of gram-negative septic_shock , although the range of organisms against which it is effective remains to be determined .
Due to its inhibitory activity for various LPS , BPI has therapeutic potential in endotoxic_shock .
These findings suggest that rBPI is a potent inhibitor of LPS-mediated responses in ECs and whole blood and underscore the potential use of BPI in treatment or prevention of endotoxic_shock .
CONCLUSIONS : This pilot study demonstrated that low ET CO2 had strong association with standard indicators for shock and was predictive of patients meeting CAT + criteria in the first 6h after admission .
The Prognostic Value of Central Venous-to-Arterial CO2 Difference/Arterial-Central Venous_O2 Difference Ratio in Septic_Shock Patients with Central Venous_O2 Saturation > = 80 .
In pediatric septic_shock patients , increasing CO2 by blood transfusion may not increase VO2 .
Among patients with both gram-negative and gram-positive septic_shock , C2_and_B concentrations were low in a subset of patients with pre-existing cirrhosis , suggesting hepatic_hyposynthesis of these proteins may be important in their prognosis and predisposition to severe infections .
Patients with septic_shock had significantly higher C3a levels than normotensive patients (p values between 0.046 and 0.004) .
In contrast to 30 healthy volunteers , 60 patients in septic_shock presented evidence of complement activation with significantly increased serum levels of C3a , C5a , and C5b-9 .
Levels of C3a and C3d were elevated in 90 % of the patients (median levels 0.19 mg/l and 8.6 mg/l respectively) in comparison to 14 % and 42 % , respectively of 7 patients with non-septic_shock .
Finally , C5ar1 - / - male mice were largely protected from histamine-induced hypovolemic_shock , which was associated with protection from histamine-induced barrier_dysfunction in vitro following C5aR targeting .
Levels of cC5aR were significantly enhanced during septic_shock , with serum levels directly correlating with lethality .
Comment on `` changes and regulation of the C5a_receptor on neutrophils during septic_shock in humans '' .
Changes and regulation of the C5a_receptor on neutrophils during septic_shock in humans .
Increased IL-1b , IL-6 , IL-8 , IL-10 , TNF-a and decreased C4 d , C5a and iC3b levels were associated with septic_shock , coma and mortality .
Neutralization of C5a with specific antibodies may reduce the hypotensive response to endotoxin sufficiently to prevent lethal septic_shock both in animals and in man .
CONCLUSION : These results suggest that excessive complement activation via C5a leads to a NHE-1-mediated shift of pHi and subsequent neutrophil functions during septic_shock .

There is evidence in sepsis , both in rodents and in humans , that activation of the complement system results in excessive production of C5a , which triggers a series of events leading to septic_shock , multiorgan_failure , and lethality .
Septic_shock patients had significantly higher plasma CGRP , and nitrite and nitrate concentrations , at each of the four time points , than patients with sepsis , as well as both groups of patients compared to normal subjects .
BACKGROUND : Cholecystokinin (CCK) , as a gastrointestinal hormone , has an important protective role against sepsis or LPS-induced endotoxic_shock .
Functional immune assays with patient Peripheral Blood Mononuclear Cells (PBMCs) revealed that burn_shock patients (> = 15 % TBSA) produced elevated levels of MCP-1 / CCL2 after innate immune stimulation ex vivo relative to mild burn patients .
Targeting CCR2 : a novel therapeutic strategy for septic_shock ?
In CD14 + monocytes of patients with septic_shock , the anti-inflammatory effect of isoprenaline was completely blunted whereas efficacy of forskolin and rolipram was maintained .
We therefore investigated whether Treg could be involved in the decreased monocyte expression of CD14 and HLA-DR observed during septic_shock .
CD30 discriminates heat_shock protein 60-induced FOXP3 + CD4 + T cells with a regulatory phenotype .
CONCLUSION : We found a major increase in the percentage of Tregs in peripheral blood circulating CD4 (+) T cells from patients with septic_shock , and observed that the removal of Tregs by hemoperfusion with PMX-F might represent a novel strategy for inducing recovery from the immunosuppression associated with sepsis .
The percentage of CD3 CD4 T lymphocytes and CD19 lymphocytes , CD4 / CD8 T cell ratio were substantially lower in patients with septic_shock compared to the control group ($P < 0.01$) .
Soluble CD40L (CD154) is increased in patients with shock .
CDD levels in plasma or serum increased from a median of 96 ng/mL in healthy control subjects to medians of 168 ng/mL in patients without persistent shock (n = 23 ; $P = .001$) and 422 ng/mL in patients with fulminant meningococcal_septicemia (n = 14 ; $P = .0001$) .
Meningococcal septic_shock in children was associated with increased serum soluble CEACAM1 .
Hyperthermia-induced heat_shock activates the transcription factor c/EBP-beta and augments IL-6 production in human intestinal epithelial cells .
COPD patients were also at significantly ($P < .05$) increased risk for any complication , such as mortality , myocardial_infarction , pneumonia , septic_shock , unplanned reintubation , use of a mechanical ventilator > 48 hours , deep_infection , require a blood transfusion , return to operating room , and a readmission within 30 days postoperatively .
All shock patients showed high levels of VIIIR : Ag , VIIIR : Cof , and VIII : CAg , averaging fivefold to sixfold the normal level , of VIII : C averaging threefold the normal level .
In contrast , CRP at admission was lower in the shock group as compared with the no-shock group (medians , 58 and 165 mg/L , respectively ; $P = 0.008$) .
Serum was analysed for the concentration of C-reactive_protein (CRP) , interleukin-6 (IL-6) , heat_shock protein 72 (eHSP72) , immunoglobulin M (IgM) and LPS .
Univariate analysis indicated that septic_shock and low C-reactive_protein (CRP) values at admission were associated with an increased risk of death .
CRP , IL-6 blood level , and mortality were significantly higher in the septic_shock group (n = 57) than in the sepsis group (n = 127) ($P < 0.001$) .
Moreover , a higher serum CRP level on the first day of admission , occurrence of bacteremia , presence of polymicrobial_infection and inadequate antibiotic use were significant risk factors for developing septic_shock .

Univariate analysis indicated that septic_shock and low C-reactive_protein (CRP) values at admission were associated with an increased risk of death .
GM-CSF as successful salvage therapy of metamizole (dipyrone) - induced agranulocytosis with Fournier 's _ gangrene and severe septic_shock in an adolescent .
Pretreatment with 10 microg of recombinant human granulocyte_colony-stimulating_factor (rhG-CSF) protected MAIDS mice from hypersensitivity to LPS-induced lethal shock and this protection was concomitant with suppression of IFN-gamma production .
When plasmas from three shock patients were depleted of native granulocyte_colony-stimulating_factor or interleukin-10 by immunoprecipitation , no increase in tumor_necrosis_factor-alpha release occurred after removal of granulocyte_colony-stimulating_factor , whereas removal of interleukin-10 increased the tumor_necrosis_factor-alpha release eight-fold .
When plasmas from three shock patients were depleted of native granulocyte_colony-stimulating_factor or interleukin-10 by immunoprecipitation , no increase in tumor_necrosis_factor-alpha release occurred after removal of granulocyte_colony-stimulating_factor , whereas removal of interleukin-10 increased the tumor_necrosis_factor-alpha release eight-fold .
Adjunctive granulocyte_colony-stimulating_factor for treatment of septic_shock due to melioidosis .
One patient with neutropenia had suspected septic_shock , which could be managed by G-CSF and antibiotics .
RESULTS : Of 1671 septic_shock patients , 158 FN patients were treated with G-CSF and 114 (72.2 %) survived for one month .
CONCLUSION : G-CSF does not improve outcomes in patients with septic_shock , excluding melioidosis .
METHODS : We performed a prospective data collection and analysis to determine whether the addition of G-CSF to our standard treatment for community-acquired septic_shock was associated with improved hospital outcome , compared with an historical cohort of similar patients .
CONCLUSION : G-CSF is a safe adjunctive therapy in community-acquired septic_shock and may be associated with improved outcome .
CysC and SCr were determined again at 72 hours and 7 days after shock .
CysC increased at 1 hour after shock .
Cathepsin_B is involved in the heat_shock induced cardiomyocytes apoptosis as well as the anti-apoptosis effect of HSP-70 .
In senescent astroglia , oxidative stress may down-regulate the cathepsin_B gene as part of a concerted cellular stress (heat_shock) response .
Nevertheless , heat_shock leads to an increase of antigen peptide generation in subcellular compartments ; an enhancement of cathepsin_B activity is also observed .
Here , we evaluate the utility of a dendritic hexadecapeptide comprised of four arms , each having a tetrapeptide sequence recognized by an enzyme cathepsin_B as a carrier system for heat_shock protein 90 (HSP90) inhibitor geldanamycin (GDM) .
The identification of parasite heat_shock proteins and annexin_A13 exclusively in infertile_cysts , along with an increased spectral count for cathepsin_B , supports the hypothesis of increased cellular stress and apoptosis as the cause of their infertility .
Proteomic expression signature composed of six biomarkers (haptoglobin , cytochrome_b5 , progesterone receptor membrane component 1 , heat_shock 27 kDa protein 1 , lysosomal proteinase cathepsin_B , keratin I) was developed as a classifier model for predicting HCC .
Enzymatically active cathepsin_B dissociating from its inhibitor complexes is elevated in blood plasma of patients with septic_shock and some malignant_tumors .

This suggests that the CC genotype of NIK rs7222094 is associated with increased mortality and organ dysfunction in septic_shock patients , perhaps due to altered regulation of NF-kB pathway genes , including CXCL10 .
Levels of IL-8 and elastase in patients with shock were significantly higher than in patients without shock (P = 0.02 ; WMW) , but those of lactoferrin were not .
Furthermore , depletion of HSF-1 using siRNA also reduced the effects HS on TNF-alpha-induced IL-8 expression , demonstrating that HSF-1 could also act to regulate IL-8 gene transcription .
TNF-alpha-induced reporter activity of an IL-8 promoter construct IL8 (-1471 / +44) - luc stably transfected in A549 cells was also enhanced by HS .
HS markedly enhanced TNF-alpha-induced IL-8 secretion in human A549 respiratory epithelial-like cells and in primary human small airway epithelial cells .
To address this hypothesis we analyzed the effect of HS on the expression of IL-8 / CXCL-8 , a member of the human CXC family of ELR (+) chemokines .
Interleukin-8 as a stratification tool for interventional trials involving pediatric septic_shock .
MCP-2 correlated with interleukin-8 , and surprisingly , with the complement activation product C3a ; these correlations further improved when analyzing patients with septic_shock or when applying gram-positive infections .
MCP-1 correlated weakly with interleukin-8 and MCP-2 , the correlations for which were most pronounced in patients with septic_shock .
Studies focused on discovery of sepsis-related biomarkers have identified interleukin-8 as a robust outcome biomarker in pediatric septic_shock .
Recent evidence suggested that interleukin-8 can be used to stratify children with septic_shock having a high likelihood of survival with standard care .
However , in contrast to the findings in pediatric septic_shock , a plasma interleukin-8 cutoff < 220 pg/mL had a negative predictive value for death of only 74 % (95 % confidence interval , 66 % to 81 %) in adults with septic_shock .
However , in contrast to the findings in pediatric septic_shock , a plasma interleukin-8 cutoff < 220 pg/mL had a negative predictive value for death of only 74 % (95 % confidence interval , 66 % to 81 %) in adults with septic_shock .
Whether plasma interleukin-8 would have similar utility in adults with septic_shock is unknown .
OBJECTIVE : Plasma interleukin-8 levels of < 220 pg/mL have an excellent negative predictive value (94 % to 95 %) for death at 28 days in children with septic_shock and thus may be useful for risk stratification in clinical trial enrollment in this population .
CONCLUSIONS : In contrast to similar pediatric patients , plasma interleukin-8 levels are not an effective risk stratification tool in older adults with septic_shock .
Plasma interleukin-8 is not an effective risk stratification tool for adults with vasopressor-dependent septic_shock .
While the production of tumor_necrosis_factor (TNF) and interleukin-6 (IL-6) in septic_shock and other inflammatory states is well established , the role of interleukin-8 (IL-8) , a recently described neutrophil chemoattractant and activator , has yet to be fully elucidated .
Effect of stress doses of hydrocortisone on S-100B vs. interleukin-8 and polymorphonuclear_elastase levels in human septic_shock .
OBJECTIVE : To examine the hypothesis that neutrophil chemotaxis to interleukin-8 (IL-8) is reduced in septic_shock .
IL-8 was demonstrated in sera from 28 of 62 patients ; levels were significantly higher in patients with septic_shock without meningitis (median , 36.1 ng/mL) than in patients with other manifestations (median , < 0.02 ng/mL) , and 4 of 5 patients who died had high levels .

There was a significant correlation between plasma IL-10 , neopterin (r = .72) , TNF-alpha (r = .76) , IL-6 (r = .68) , and IL-8 (r = .61) levels in patients with septic_shock .
These results indicate that IL-8 and PMNE are produced in large quantities when septic_shock occurs , and may play a role in the development of septic ARDS .
We determined the plasma concentrations of interleukin_8 (IL-8) , polymorphonuclear leukocyte_elastase (PMNE) , and endotoxin in patients with septic_shock in order to investigate the role of IL-8 and PMNE in the development of septic_shock , especially in septic adult_respiratory_distress_syndrome (ARDS) .
Increased IL-1b , IL-6 , IL-8 , IL-10 , TNF-a and decreased C4 d , C5a and iC3b levels were associated with septic_shock , coma and mortality .
Patients with septic_shock showed higher levels of IL-8 , GM-CSF , MIP-1b than those with SIRS .
Plasma levels of cytokines , including interleukin_6 (IL-6) , IL-8 , IL-10 , and high-mobility_group_box_1 , were elevated in patients with septic_shock compared with healthy controls , but cytokine levels were not altered by PMX-DHP .
Here we report that IL-8 , a novel cytokine produced by a variety of cells in vitro in response to stimulation with bacterial LPS and the proinflammatory cytokines , appears in the circulation of primates in vivo during septic_shock , sublethal endotoxemia , and after the administration of IL-1_alpha .
While the production of tumor_necrosis_factor (TNF) and interleukin-6 (IL-6) in septic_shock and other inflammatory states is well established , the role of interleukin-8 (IL-8) , a recently described neutrophil chemoattractant and activator , has yet to be fully elucidated .
CONCLUSIONS : A serum IL-8 level of 220 pg/ml or less , obtained within 24 hours of admission , predicts a high likelihood of survival in children with septic_shock .
A serum IL-8 level of 220 pg/ml or less , however , had a negative predictive value for 28-day mortality of 95 % in validation data set 1 , which was subsequently applied to an independently generated data set of children with septic_shock (validation set 2 , n = 193) .
RESULTS : Concentrations of IL-1_beta , IL-6 , IL-7 , IL-8 , IL-10 , IL-13 , interferon-gamma , MCP-1_and_tumour_necrosis_factor-alpha were significantly higher in septic_shock patients than in those with severe sepsis .
IL-8 , a cytokine known for its potent and specific neutrophil activation and chemoattractant properties , has been recently detected in the circulation during septic_shock , endotoxemia , and after IL-1_alpha administration .
High blood levels of IL-8 have been found in patients with septic_shock .
OBJECTIVE : To examine the hypothesis that neutrophil chemotaxis to interleukin-8 (IL-8) is reduced in septic_shock .
Evidence for a protective role for the rs805305 single nucleotide polymorphism of dimethylarginine_dimethylaminohydrolase_2 (DDAH2) in septic_shock through the regulation of DDAH activity .
Although L-NAME does not increase ET-1 concentration in patients with septic_shock , the vasopressor response induced by L-NAME depends on the plasma level of ET-1 .
Although L-NAME does not increase ET-1 concentration in patients with septic_shock , the vasopressor response induced by L-NAME depends on the plasma level of ET-1 .
These results may mean that the level of the concentration of ET-1 plays a key role for prevention of the multiple_organ_failure even after the recovery from septic_shock .
When patients recovered from the septic_shock , plasma ET-1 levels rapidly decreased .
In cases with good prognosis after the septic_shock , ET-1 was significantly higher as compared with these in sepsis without shock .

Elevated plasma levels of endothelin-1 (ET-1) in patients with septic_shock have implicated ET-1 in the vascular hypoperfusion and organ_dysfunction , including renal_failure , that occur in this condition .
Our data suggest that ET-1 may be associated with septic_shock in patients undergoing hemodialysis and that PMX-F is effective in reducing plasma ET-1 levels in these patients .
Endothelin-1 and blood pressure after inhibition of nitric_oxide synthesis in human septic_shock .
Endothelin-1 and atrial natriuretic peptide in septic_shock .
Elevated plasma levels of endothelin-1 (ET-1) in patients with septic_shock have implicated ET-1 in the vascular hypoperfusion and organ_dysfunction , including renal_failure , that occur in this condition .
[The role of endothelin-1 in the pathogenesis of septic_shock in the abdominal_sepsis] .
From the PPI network , the top 10 hub genes , which are all upregulated DEGs in the septic_shock children , were identified as GAPDH , TNF , EGF , MAPK3 , IL-10 , TLR4 , MAPK14 , IL-1b , PIK3CB , and TLR2 .
Since receptor dimers have been implicated in the EGF-induced activation of EGF_receptor , hyperosmotic_shock may activate EGF_receptor by a different mechanism .
Plasma interleukin_8 and polymorphonuclear leukocyte_elastase concentrations in patients with septic_shock .
In contrast , patients with septic_shock presented 1.5-fold higher levels of endoglin (P = 0.004) , and 2-fold lower levels of Heparin-Binding_EGF-like_growth_factor (HB-EGF) (P = 0.002) when compared to healthy individuals .
In this study , we examined the effects of heat_shock on the expression of recombinant human erythropoietin (EPO) in a Chinese_hamster ovary (CHO) cell line .
Changes in plasma erythropoietin and interleukin-6 concentrations in patients with septic_shock after hemoperfusion with polymyxin B-immobilized fiber .
Similar to interleukin_6 , abnormally high serum erythropoietin levels appear to be a negative prognostic indicator in patients suffering from septic_shock .
This editorial comment discusses the effects of erythropoietin in preclinical models of septic_shock , endotoxemia , hemorrhagic_shock , spinal_cord_trauma and zymosan-induced multiple_organ_failure .
This relationship could suggest tissue hypoperfusion as the stimulating factor for erythropoietin production in septic_shock .
High serum erythropoietin levels in non-survivors were observed with septic_shock despite an increase in the levels of proinflammatory cytokines .
Serum erythropoietin levels in septic_shock .
Endocan levels were higher in patients with septic_shock (6.11 + / - 12.99 ng/mL , n = 22) than in patients with severe sepsis (1.97 + / - 7.8 ng/mL , n = 12) or sepsis (1.95 + / - 1.63 ng/mL , n = 29) .
Tissue_factor (TF) plays a critical role in the pathogenesis of disseminated intravascular_coagulation (DIC) observed in patients with septic_shock .
Cellular immune and cytokine pathways resulting in tissue_factor expression and relevance to septic_shock .
During Gram-negative septic_shock , lipopolysaccharide (LPS , endotoxin) induces tissue_factor (TF) expression .
We aimed to determine retrospectively whether urinary liver-type_fatty_acid-binding_protein (L-FABP) levels are altered in patients with septic_shock or severe_sepsis without shock and whether polymyxin B-immobilized fiber (PMX-F) hemoperfusion affects these levels .
These results suggest that urinary L-FABP levels are significantly increased in patients with septic_shock and that PMX-F treatment is effective in reducing these levels .

The serum level of I-FABP in the septic_shock group was significantly higher than that in the sepsis group (P < 0.05) , but the difference in serum D-lactic_acid level between the two groups was not statistically significant (P > 0.05) .
Neutrophil CD64 (nCD64) expression was higher in patients with SS (81.2 %) and S (78.8 %) as compared to those with TBI (5.5 %) or C (0.9 % , p < 0.0001) .
CD64 at day 1 was higher with patients with septic_shock when compared with sepsis (P = 0.012) .
While fibroblast_growth_factor_23 (FGF-23) has recently emerged as a promising predictor of mortality in patients with cardiogenic_shock , its role in risk stratification in post-resuscitation management remains unresolved .
In patients with CS , FGF-23 correlated significantly with the SAPS II score (r = 0.461 , p = 0.0003) and NT-pro BNP levels (r = 0.489 , p = 0.001) .
Compared with patients with stable CAD , FGF-23 was profoundly elevated in patients with CS , but not in patients with uncomplicated AMI (CAD : 131.1 9.5 ; AMI : 175.3 57.2 ; CS : 1684.4 591.7 rU/ml , p < 0.0001 CS vs. CAD) .
METHODS AND RESULTS : FGF-23 was measured in 51 patients with CS .
CONCLUSION : In CS , a tremendous increase in FGF-23 occurs , and high levels of FGF-23 are associated with poor outcome .
While fibroblast_growth_factor_23 (FGF-23) has recently emerged as a promising predictor of mortality in patients with cardiogenic_shock , its role in risk stratification in post-resuscitation management remains unresolved .
FGF-23 is associated with increased_disease severity and early mortality in cardiogenic_shock .
Plasma fibronectin (FNp) concentrations were measured in 63 patients with acute_respiratory_failure and 28 patients with circulatory_failure , using Laurell 's electroimmunoassay method .
[Prognostic value of plasma levels of fibronectin in septic_shock] .
In fourteen pigs aortic surgery produced reproducible surgical_shock and a fall in plasma fibronectin from 331 + / - 10 mg/l to 43 + / - 13 mg/l after resuscitation (P less than 0.01) .
Furthermore we show that casein kinase II , which has been proposed to be involved in serum induction via the serum response element , may also be involved in heat_shock , arsenite and cadmium induction of c-fos .
CD30 discriminates heat_shock protein 60-induced FOXP3 + CD4 + T cells with a regulatory phenotype .
Levels of Growth_Differentiation_Factor_15 and Early Mortality Risk Stratification in Cardiogenic_Shock .
METHODS AND RESULTS : Levels of GDF-15 were determined in serial plasma samples (0-120 h) from 177 CS patients in the CardShock study .
CONCLUSIONS : GDF-15 levels are highly elevated in CS and associated with markers of systemic_hypoperfusion_and_end-organ_dysfunction .
Enhanced growth_hormone response to clonidine after repeated electroconvulsive_shock in a primate species .
AIM : To investigate the therapeutic effects of recombinant human growth_hormone (rhGH) on rat septic_shock with intraabdominal_infection by E. _ coli and its possible mechanism .
Effects of recombinant human growth_hormone on rat septic_shock with intraabdominal_infection by E. _ coli .
In man , both PGI_and_TxB were significantly increased in severe sepsis , compared to normal controls , but only PGI was significantly higher in septic_shock versus normotensive sepsis .
Heat_shock of insulin-treated cultures causes induction of the 82K GRP rather than the 85K and 69K HSP 's .

RESULTS : GRP markly increased the relative promoter activity of hsp90alpha-CAT reporter gene during heat_shock .
Following hemorrhagic_shock , gelsolin levels decrease significantly , possibly due to consumption by the actin scavenging system .
Decreased plasma gelsolin concentrations in acute_liver_failure , myocardial_infarction , septic_shock , and myonecrosis .
Patients with septic_shock had higher HBb levels when compared to patients with severe sepsis .
The results revealed that Tiam1 transfection in colorectal_cancer cells could upregulate the expression of Fascin-1 , heat_shock protein 27 (HSP27) , high-mobility_group_box_1 (HMGB1) , glutathione_S-transferase omega 1 (GSTO1) and downregulate the expression of annexin IV .
High-mobility_group_box_1 (HMGB1) and receptor for advanced glycation end-products (RAGE) axis is also involved in septic_shock .
High-mobility_group_box_1 (HMGB1) and receptor for advanced glycation end-products (RAGE) axis is also involved in septic_shock .
OBJECTIVE : To investigate plasma high-mobility_group_box_1 protein (HMGB1) concentration and its relationship with organ_dysfunction and outcome in septic_shock patients .
Expression of HO-1 in vivo suppresses the inflammatory responses in endotoxic_shock , hyperoxia , acute_pleurisy , and organ transplantation , as well as ischemia-reperfusion injury , and thereby provides salutary effects in these conditions .
The objective of this study was to improve the role of HO-1 on systemic inflammatory response in an endotoxic_shock model .
The first results of therapeutic intervention with C1-INH concentrate in septic_shock are promising .
Induction of HSP90_alpha heat_shock mRNA after transient global ischemia in gerbil hippocampus .
The results suggest that in addition to HSP90 in Ag donor cells , endogenous HSP90 in DCs plays an essential role during Ag cross-presentation and , moreover , points to a link between heat_shock factor-1-dependent induction of HSP90alpha within DC and cytotoxic T cell immunity .
A single nucleotide polymorphism (SNP) that causes a missense mutation of highly conserved Gln488_to_His of the alpha isoform of the 90-kDa heat_shock protein (Hsp90alpha) molecular chaperone is observed in Caucasians .
Heat_shock and antioxidants induced Hsp90 , and casein kinase 2 (CK2) phosphorylated INrf2Thr55 .
We developed a four-colour single-molecule FRET system to observe the succession of states in the heat_shock protein 90 (Hsp90) system , consisting of an Hsp90 dimer , the cochaperone p23 and nucleotides .
Association with hsp90 and hsp70 heat_shock proteins .
CONCLUSIONS : Rac-MEKK-JNK pathway promotes heat_shock induced hsp90_beta gene expression and hsp90 may participate in the regulation of heat_shock activated Rac-MEKK-JNK signal pathway in both Jurkat and LETPa-2 cells .
Therefore , phosphorylation and interaction with hsp90 both contribute to stabilization of p53 after heat_shock .
Heat_shock (42 degrees C) markedly increased the synthesis of proteins with apparent molecular weights of 98 , 89 , 72 , 50 , 42 and 25 KDa , with HSP_89 and 72 being most prominent .
Heat_shock induction of HSP_89 is regulated in cellular aging .
Heat_shock induced HSP-72 production , which was dependent on both temperature and the duration of heating .
HSP72 mRNA and l/mHSP72 are higher among critically_ill patients , further induced by HS , not by LPS .

The content of HSP72 induced by HS also correlated well with the induction , release , and maintenance of G1_arrest .
On the other hand , HS induced HSP72 expression markedly .
On the other hand , activating the HSR increases heat_shock protein 72 (HSP72) expression and improves insulin resistance and glucose homeostasis in rodents and humans , possibly by inhibiting the activation of stress kinases such as c-jun terminal kinase (JNK) and inhibitor of kappa B kinase b (IKKb) .
In addition , after HA , the Hsp72 response to HS was reduced (day 1 , 129 46 ; day 10 , 80 32 ng mL (-1) , $p < 0.05$) .
HS treatment caused a greater increase ($p < 0.05$) in Hsp72 than the exercise sessions on HA days 1 and 10 .
Geldanamycin (GA) , a specific inhibitor of the chaperoning function of heat-shock_protein_90 (Hsp90) , has been shown to mimic heat_shock (HS) in inducing expression of Hsp90 , Hsp72 and other Hsps in unstressed mammalian cells .
This process may involve increased expression of heat_shock proteins , in particular the highly inducible heat_shock protein 72 (Hsp72) .
Heat_shock at 42 degrees C for 30 min increased hsp72 levels but caused no change in hsp90 .
For hsp72 (which is reproducibly induced in all three cell lines to relatively high levels of expression) , we studied U937 cells before and after heat_shock .
In contrast , heat_shock (both acute and chronic) led to a non-uniform increase in hsp72 through the cell cycle .
The response to cellular stress (e.g. hyperthermia) in somatic cells includes activation of the heat_shock transcription factor 1 (HSF1) leading to induction and accumulation of the heat_shock proteins (HSPs) , mainly HSP70i , which allows cell survival .
RESULTS : HS induced long-lasting expression of Hsp70/72 and Hsp90 .
Our study was designed to determine whether heat_shock and drugs like cisplatin , etoposide and quercetin influence the expression of heat_shock_protein_72 in tumour cells : HeLa (cervical_cancer) , Hep-2 (larynx_cancer) , A549 (lung cancer) and normal human skin fibroblasts (HSF) .
Results : Forty-nine percent of the AD patients showed increased IgG-reactivity to any of the four antigens representing keratin_associated_protein_17-1 (KRTAP17-1) , heat_shock protein family A (Hsp70) member 4 (HSPA4) , S100 calcium binding proteins A12 (S100A12) , and Z (S100Z) .
RESULTS : HS induced long-lasting expression of Hsp70/72 and Hsp90 .
Increases in HSF1 translocation and synthesis in human epidermoid A-431 cells : role of protein_kinase_C and [Ca2 +] i. BACKGROUND : It is known that heat_shock increases both heat_shock protein 70 kd (HSP-70) mRNA synthesis , and intracellular cytosolic free calcium concentration ([Ca2 +] i) .
Flow cytometry showed increased levels of HSP_70 and 90 after heat_shock at 41.8 degrees C for 60 minutes , measured after a subsequent incubation time of five hours , as compared to untreated cells in vitro .
We have previously described the induction of heat_shock (HS) protein (HSP) (in particular , HSP70) in human monocytes exposed to TS .
These results demonstrate that in glial cells , as well as other cell types , NOS-2 induction can be modulated by the HS response , mediated at least in part by HSP70 expression .
Interestingly , the enhancing effect of HS was partially inhibited by the addition of the heat_shock_protein_70 (HSP70) - inhibitor pifithlin - (PFT) .
HS preconditioning (1 h at 42 C) induced a rapid increase in HSPA1 (HSP70) levels which remained elevated for at least 48 h post-HS .

The heat_shock_protein_70 (HSP70) was significantly upregulated by HS in differentiated hMSCs analyzed at 24 h after HS .
The heat_shock_protein_70 (HSP70) was significantly upregulated by HS in differentiated hMSCs analyzed at 24 h after HS .
The results showed that the expression of HSP70 in the HS2h oocytes was higher ($p < 0.05$) than those had recovery incubation for 1 h (HC1h) after HS , but the cleavage and blastocyst rates were greater ($p < 0.05$) in the HC1h group .
Combination of HS and radiation treatment significantly induced the transcription of the HSP70 gene above the level induced by each stressor alone .
The ubiquitous heat_shock (HS) protein HSP70 , expressed under the control of the heat_shock transcription factor 1 (HSF-1) , is recognized as one of the main chaperones associated with cell protection against stresses .
The results clearly show that HS genes , in particular the three HSP70 genes (A , B , and C) , are induced by ELF-EMF , a reaction that is enhanced by simultaneous HS (43 degrees C for 30 min) .
The results clearly show that HS genes , in particular the three HSP70 genes (A , B , and C) , are induced by ELF-EMF , a reaction that is enhanced by simultaneous HS (43 degrees C for 30 min) .
By contrast , isoproterenol plus theophylline failed to attenuate the stimulation of HSP70 gene expression and HSF binding activity caused by heat-shock .
Constitutive expression of HSP27 , HSP70 , HSC70 , HSP90alphabeta and GRP94 proteins was found in all the melanoma cell lines , and HSP70 and HSC70 were also induced by heat_shock .
In this study , we report that the calcium ionophore A23187 , a glucose-regulated_protein (GRP) inducer , dramatically inhibits HSP70 synthesis and HSP70 mRNA transcription after induction by heat_shock , sodium_arsenite , or prostaglandin_A1 treatment in human K562 cells .
It is likely that in the absence of Hikeshi , HSP70 can not attenuate the multiple heat_shock induced nuclear phenotypes , leaving the cells unprotected during heat_shock stress .
Food odor , visual danger stimulus , and retrieval of an aversive memory trigger heat_shock protein HSP70 expression in the olfactory lobe of the crab Chasmagnathus granulatus .
It is concluded that the heat_shock and ADM chemotherapy both induce over expression of HSP70 and MDR1 which can maintain stability of K562 cells and may be related to formation of the MDR in leukemia .
The results showed that heat_shock increased the expression of HSP70 and HSP90 of HELAs , while cold_shock decreased the expression of the two proteins .
We autopsied two MA-detected cadavers , and immunohistochemical staining was performed on the skeletal muscle with an anti-myoglobin antibody , and on the kidney with an anti-the 70 kDa heat_shock protein (HSP70) antibody .
HSP70 and HSP90 are induced 3 h after release from heat_shock , whereas HSP27 is induced much later .
Involvement of a chloroplast HSP70 heat_shock protein in the integration of a protein (light-harvesting complex protein precursor) into the thylakoid membrane .
We found that mild heat_shock upregulates not only HSP70 but also BCL-2 , though BCL-2 has not previously been recognized as a heat-inducible protein .
HSP70 expression was inducible by thermal heat_shock in the PBMC of both patients and healthy individuals .
However , skeletal muscle mRNA of cold_shock proteins decrease , while HSP70 mRNA increases in response to a low to moderate intensity aerobic exercise bout .
The results showed that heat_shock increased the expression of HSP70 and HSP90 of HELAs , while cold_shock decreased the expression of the two proteins .

From the results of this study , we conclude that the expressions of HSP70 and HSP90 in HELAs increased significantly after heat_shock , while cold_shock decreased the expressions of these two proteins .
We demonstrate that without heat_shock , the levels of the inflammatory mediators are positively related to Hsp_70 production in monocytes .
We determined whether Hsp70 induction by TS was mediated by the activation of the HS transcription factor , HSF .
Expression of Hsp70 was not uniform within the monocyte population , indicating the presence of subpopulations expressing variable levels of Hsp70 in response to HS .
Higher relative fluorescence intensity was observed in cells exposed to heat_shock (HS) , reflecting a higher expression of Hsp70 in these cells as compared with cells kept at 37 degrees C .
We chose pairs of mammalian and insect species that significantly differ in their thermoresistance and constitutive levels of Hsp70 to compare hsp promoter strength under normal conditions and after heat_shock (HS) .
In parallel , HS increased both Hsp70 and Mcl-1 protein levels in PMNL .
Using immunoblotting with human recombinant Hsps and univariate and multivariate logistic regression models , we have investigated the presence of antibodies against Hsp70 , the inducible member of the 70-kDa family of heat_shock_proteins , and analyzed its possible association with hypertension and working conditions .
Heat_shock was the strongest inducer of Hsp70 and Hsp90 .
Marked differences between avian and mammalian testicular cells in the heat_shock induction and polyadenylation of Hsp70 and ubiquitin transcripts .
Treatment with sublethal heat_shock or TNF-alpha results in the up-regulation of intracellular Hsp70 in FLSs and Hsp70 release from RA FLSs .
Thus , these results lend support to the hypothesis that Hsp70 is actively released from FLSs in response to heat_shock or TNF-alpha and Hsp70 may be a major paracrine/autocrine inducer of IL-10 production in FLSs via TLR4 .
The cells secreted IL-8 in response to S. serovar Enteritidis and produced Hsp70 after heat_shock or incubation with butyrate .
On heat_shock , Hsp70 is rapidly recruited to mitotic centrosomes and normal progression through mitosis is observed immediately after release of Hsp70 from centrosomes .
When levels of Hsp70 are elevated after heat_shock , or in cells conditionally overexpressing Hsp70 , Bag1-Raf-1 is displaced by Bag1-Hsp70 , and DNA synthesis is arrested .
is not involved in the PMA-mediated induction of hsp70 and hsp90 and that , in contrast to HS , PMA increases the expression of HSP as a result of PKC-induced mRNA stabilization rather than of transcriptional activation of HS genes .
Our results showed that HS treatment increased the expression of the hsp70 protein and protected the cells from apoptosis in vitro .
Heat_shock induces synthesis of plastid-associated hsp70 in etiolated and greening pumpkin seedlings .
Further , heat_shock of parental HL-60 cells at 42 degrees C for 3 h increased hsp70 levels , promoted plastic adherence (< 6 h) of the cells in respond to PMA , and protected cells from SNP or Taxol .
Deficient induction of human hsp70 heat_shock gene transcription in Y79 retinoblastoma cells despite activation of heat_shock factor 1 .
After heat_shock , hsp70 binds tightly first to some nuclear component (s) and then to nucleoli .
The objectives of this study were to determine the ability of trophectoderm from preimplantation ovine embryos to synthesize hsp70 in response to heat_shock and to identify conditions which induce translational thermotolerance in this tissue .

Given the newly described cell signaling properties of hsp70 , these data suggest that extracellular hsp70 may play a role in the host response during septic_shock .
Extracellular hsp70 levels in children with septic_shock were significantly elevated compared with control patients (51.6 ng/mL vs. 8.1 ng/mL , respectively , p = .0004) .
BACKGROUND AND PURPOSE : Previously , we demonstrated that exogenous heat_shock protein 27 (HSP27/gene , HSPB1) treatment of human endothelial progenitor cells (EPCs) increases the synthesis and secretion of VEGF , improves EPC-migration/re-endothelialization and decreases neo-intima formation , suggesting a role for HSPB1 in regulating EPC function .
Heat_Shock Protein HSP27 Secretion by Ovarian_Cancer Cells Is Linked to Intracellular Expression Levels , Occurs Independently of the Endoplasmic Reticulum Pathway and HSP27 's Phosphorylation Status , and Is Mediated by Exosome Liberation .
Heat_shock , however , causes an increase in HSP27 when HSF1 is up-regulated , except when the expression of HSF1 is already too high .
The level of protection provided by HSP27 during heat_shock may thus represent the contribution of better maintenance of actin filament integrity to overall cell survival .
Metastatic and/or primary tumour tissue was stained by immunohistochemistry for selected markers related to angiogenesis [vascular_endothelial_growth_factor_A (VEGF-A) , VEGF_receptor_2 (VEGFR2) , platelet-derived_growth_factor_receptor_b (PDGFRb) , and heat_shock protein 27 (HSP27)] and immune responses [Interleukin_6_receptor_a (IL6Ra) , interleukin-6 (IL6) , and jagged1 (JAG1)] .
The cellular HSP27 concentration is not increased appreciably at 2 h after heat_shock and attains a maximum at 14 h. Similar results were obtained in the case of another heat_shock protein , HSP70 .
It has been previously reported that , following heat_shock , HSP27 binds to the insoluble granules of eIF4G and impedes its association with cytoplasmic poly (A) - binding_protein (PABP) 1 and eIF4E .
The basal levels and also the levels of Hsp27 production after HS were higher for monocytes compared to lymphocytes .
Distinct effects of heat_shock and ATP depletion on distribution and isoform patterns of human Hsp27 in endothelial cells .
Hsp27 levels also increased after heat_shock , but only in NC cells .
Hsp27 and actin showed colocalization before heat_shock , little association 3 h after heat_shock , and increased association 24 h after heat_shock .
Constitutive expression of human hsp27 resulted in a 100-fold increase in survival to a single lethal heat_shock in CHO cells without effecting the development of thermotolerance .
Previously we demonstrated that heat_shock protein 27 (hsp27) overexpression confers resistance to the chemotherapeutic agent doxorubicin in MDA-MB-231 breast_cancer cells .
However , quantification of NGF-induced neurite elongation and branching revealed that neither of these features were altered in PC12 cells which stably overexpressed human Hsp27 (to mimic heat_shock induction of Hsp25) .
More recently , heat_shock proteins (HSP) , particularly heat_shock protein 60 (HSP60) , have receive increasing attention as another possible factor in the pathogenesis and development of acute pancreatitis .
Different classes of molecular chaperones , such as the members of the Hsp70 and Hsp60 families of heat-shock_proteins , cooperate in a coordinated pathway of cellular protein folding .
Heat_shock induced the expression of Hsp60 and Hsp70 but not of adhesion molecules .
RESULTS : In SW13 cell , hsp70 and hsp90_alpha were typical heat_shock induced genes , while hsp60 and hsp90_beta were efficiently expressed and further induced by heat-shock to various extent .
A pretreatment with cadmium , which hardly induces synthesis of this hsp , does induce a tolerance to (re) - induction by heat_shock , which normally induces hsp60 .

RESULTS : Extracellular Hsp60 levels were significantly higher in children with septic_shock (median , 16.7 ng/mL) compared to both critically ill children without septic_shock (median , 0 ng/mL) and healthy controls (median , 0 ng/mL , p < 0.001) .
Heat_shock inhibits tnf-induced ICAM-1 expression in human endothelial cells via I kappa kinase inhibition .
We conclude that heat_shock decreases endothelial cell ICAM-1 expression via inhibition of IKK activity .
IFN-gamma was maintained in a folding-competent form by calreticulin during heat-shock and released during subsequent recovery at 37 degrees C .
RESULTS : Concentrations of IL-1_beta , IL-6 , IL-7 , IL-8 , IL-10 , IL-13 , interferon-gamma , MCP-1_and_tumour_necrosis_factor-alpha were significantly higher in septic_shock patients than in those with severe sepsis .
In addition , it appears as if interferon-gamma plays a minor role in the pathophysiology of meningococcal septic_shock .
When analyzed for AKI etiology , highest [TIMP-2] [IGFBP7] values were found in patients with septic_shock (P < 0.001 vs. non-AKI I+II) .
Most bacterial_endotoxin_shock patients had lower levels of serum IL-10 than cardiogenic_shock patients .
Patients with septic_shock (n = 21) had higher interleukin-10 (main 58 pg/mL) than septicaemic patients without shock (11 pg/mL , p < 0.001) .
Role of interleukin-10 in monocyte hyporesponsiveness associated with septic_shock .
We conclude that IL-10 is extensively activated along with the proinflammatory cytokines during the initial phase of meningococcal septic_shock and that IL-10 is associated with fatality in meningococcal_disease .
Increased IL-1b , IL-6 , IL-8 , IL-10 , TNF-a and decreased C4 d , C5a and iC3b levels were associated with septic_shock , coma and mortality .
From the PPI network , the top 10 hub genes , which are all upregulated DEGs in the septic_shock children , were identified as GAPDH , TNF , EGF , MAPK3 , IL-10 , TLR4 , MAPK14 , IL-1b , PIK3CB , and TLR2 .
RESULTS : Concentrations of IL-1_beta , IL-6 , IL-7 , IL-8 , IL-10 , IL-13 , interferon-gamma , MCP-1_and_tumour_necrosis_factor-alpha were significantly higher in septic_shock patients than in those with severe sepsis .
The continued release of IL-10 may contribute to impairment of monocyte proinflammatory cytokine release and the development of immune_dysfunction in septic_shock .
CONCLUSION : Monocytes from patients with septic_shock exhibit persistent IL-10 release at a time when TNF-alpha release is downregulated .
RESULTS : Concentrations of IL-1_beta , IL-6 , IL-7 , IL-8 , IL-10 , IL-13 , interferon-gamma , MCP-1_and_tumour_necrosis_factor-alpha were significantly higher in septic_shock patients than in those with severe sepsis .
IL-17 was released at greater amounts from PBMCs of non-survivors by septic_shock and AKI but not of septic_shock and CRD .
RESULTS : The plasma IL-18 levels were significantly higher in septic_shock patients (1,320 + / - 360 pg/ml) than in healthy volunteers (140 + / - 60 pg/ml ; p < 0.001) .
CONCLUSIONS : IL-18 may be associated with the severity of septic_shock , and PMX-F treatment is effective in reducing the IL-18 level in patients with septic_shock .
Interleukin-1 receptor antagonist reduces mortality from endotoxin shock .

We now report that a specific interleukin-1 receptor antagonist reduces the lethality of endotoxin-induced shock in rabbits , indicating that interleukin-1 does indeed play an important part in endotoxin shock .
The cytokine interleukin-1 has been implicated as an important mediator of septic_shock because it can induce tachycardia and hypotension and act synergistically with tumour necrosis factor to cause tissue_damage and death .
Interleukin-1alpha (IL-1alpha) , LPS and TNF-alpha , which are all known to be elevated in septic_shock , were used as stimulators at concentrations commonly found in patients with sepsis .
However , studies with a recombinant human interleukin-1 _ (IL-1) _ receptor_antagonist_protein (IL-1ra) suggest a role for IL-1 as a mediator of septic_shock as well .
IL-1 was implicated as a cardiodepressant factor in septic_shock , and subsequent pre-clinical and clinical research has defined important roles for IL-1 in atherosclerosis , acute_myocardial_infarction (AMI) , and heart_failure (HF) .
RESULTS : Median levels of interleukin-1_beta were higher in response to heat_shock protein in cultures from patients with vulvar_vestibulitis_syndrome (median , 1.07 ng/mL) as opposed to control subjects (median , 0.40 ng/mL ; P = .006) .
Interestingly , IL-1beta mRNA is elevated in hippocampus 4 h after IS , but an increase of IL-1beta protein in hippocampus is not detected .
IS increases IL-1beta mRNA and/or protein in a variety of tissues , including hypothalamus , hippocampus , pituitary and spleen .
In other experiments , IL-1beta (0.5 ng/ml) was added following heat_shock and recovery .
IL-1b and IL-2 did not change significantly with ESWT .
Interleukin-1beta (IL-1beta) is considered an important mediator in the pathogenesis of septic_shock or bacterial_meningitis .
CONCLUSIONS : Adjunctive therapy with anti-ovine interleukin-1beta monoclonal antibody in ovine gram-negative septic_shock was associated with improved hemodynamic performance .
Although IL-1_beta was never found , TNF_alpha was most often observed in the serum at a level under 100 pg/mL except during septic_shock .
Interleukin_1_beta (IL-1_beta) , tumor_necrosis_factor (TNF_alpha) , and interleukin_6 (IL-6) were measured each day and every 1 or 2 hours when septic_shock occurred .
In early septic_shock , chronic alcoholic patients had significantly decreased levels of IL-1beta ($P < 0.015$) , IL-6 ($P < 0.016$) and IL-8 ($P < 0.010$) .
Interestingly , epinephrine suppressed the IL-1beta production by 73 % ($P < 0.0001$) in blood of patients in prolonged septic_shock , which was twice as much as in blood samples of healthy volunteers .
From the PPI network , the top 10 hub genes , which are all upregulated DEGs in the septic_shock children , were identified as GAPDH , TNF , EGF , MAPK3 , IL-10 , TLR4 , MAPK14 , IL-1b , PIK3CB , and TLR2 .
The administration of IL-1ra blocks the effects of IL-1 in some animal models of septic_shock , inflammatory_arthritis , graft-versus-host_disease , and inflammatory_bowel_disease .
It may be that genetically determined IL1RA levels influence survival from septic_shock .
Recombinant IL-1Ra (anakinra) has been administered to over 1,000 patients with septic_shock resulting in a consistent reduction in all-cause 28-day mortality .
In a Phase III trial , IL-1ra reduced mortality rate in patients with septic_shock_syndrome by 22 % .
A dose-related survival benefit was observed with infusion of IL-1ra in patients with septic_shock at study entry ($n = 65$; $p = .002$) and in patients with Gram-negative infection ($n = 45$; $p = .04$) .
OBJECTIVE : To evaluate the role of NG-methyl-L-arginine as a modulator of the hyperdynamic_shock induced by the administration of interleukin-2 (IL-2) .

We defined two types of septic_shock from these data , i.e. , endotoxin + TNF-alpha + IL-2 shock and IL-beta + IL-6 shock .
These studies demonstrate widespread hemodynamic and vascular effects of IL-2 administration that limit its safe use and suggest a possible role for the lymphokine in mediating cardiovascular instability under other circumstances , such as endotoxic_shock .
IL-37 plays a role in protecting the body against endotoxin_shock , ischemia-reperfusion injury , autoimmune_diseases , and cardiovascular_diseases .
The human cytokine interleukin (IL) -37 has potent anti-inflammatory capacities , and hematopoietic cell-specific transgenic overexpression of IL-37 in mice protects against septic_shock_and_colitis .
Interleukin-6 production in human intestinal epithelial cells increases in association with the heat_shock response .
Similar to previous observations , we find that burn_shock patients (> = 15 % Total Burn Surface Area (TBSA) injury) have elevated levels of the innate immune cytokines Interleukin-6 (IL-6) and Monocyte_Chemoattractant_Protein-1 (MCP-1) / CC-motif_Chemokine_Ligand_2 (CCL2) early after hospital admission (0-48 Hours Post-hospital Admission (HPA) .
Heat_shock also increased lipoteichoic_acid - or lipopolysaccharide-induced interleukin-6 production by monocytes .
Whereas a fever-induced heat_shock response could affect expression of acute-phase proteins in the liver , the effects of a modest temperature increase on protein secretion in interleukin-6 (IL-6) - stimulated HepG2 cells were investigated .
Treatment of human peripheral blood lymphocytes (PBL) in vitro with the cytokine interleukin-6 (IL-6) induces increased levels of the 90 kDa heat_shock protein (hsp90) .
Venous blood was obtained prior to , immediately post and 2 h post-session for assessment of monocyte intracellular heat_shock protein 72 (iHsp72) and plasma concentrations of extracelullar heat_shock protein 72 (eHsp72) , interleukin-6 (IL-6) , fasting glucose , insulin and nitrite .
Several factors contribute to the exercise-induced heart preconditioning , among which the most important can be : increased activity of the anti-radical defense system , opioids , interleukin-6 , nitric_oxide , ATP dependent potassium channels , heat_shock protein 72 and sphingosine-1-phosphate .
Metastatic and/or primary tumour tissue was stained by immunohistochemistry for selected markers related to angiogenesis [vascular_endothelial_growth_factor_A (VEGF-A) , VEGF_receptor_2 (VEGFR2) , platelet-derived_growth_factor_receptor_b (PDGFRb) , and heat_shock protein 27 (HSP27)] and immune responses [Interleukin_6_receptor_a (IL6Ra) , interleukin-6 (IL6) , and jagged1 (JAG1)] .
Serum was analysed for the concentration of C-reactive_protein (CRP) , interleukin-6 (IL-6) , heat_shock protein 72 (eHSP72) , immunoglobulin M (IgM) and LPS .
Furthermore , plasma heat_shock protein 70 levels were negatively correlated with ex vivo production of inflammatory mediators interleukin-6 , tumor_necrosis_factor-a , and interleukin-10 .
Phosphorylation status of heat_shock protein 27 regulates the interleukin-1b-induced interleukin-6 synthesis in C6 glioma cells .
In particular , lactate up-regulates the expression of interleukin-6 (3 days , 4.11-fold) , of heat_shock protein 70 (3 days , 2.36-fold) and of hypoxia-inducible_factor-1alpha (3 days , 2.09-fold) .
Small heat_shock proteins associated with cerebral_amyloid_angiopathy of hereditary_cerebral_hemorrhage with amyloidosis (Dutch type) induce interleukin-6 secretion .
Febrile-range temperature but not heat_shock augments the acute phase response to interleukin-6 in human hepatoma cells .
Furthermore , heat_shock increased the synthesis and release of interleukin-6 (IL-6) into culture media .

Effect of transforming_growth_factor-beta1 , interleukin-6 , and interferon-gamma on the expression of type I collagen , heat_shock protein 47 , matrix_metalloproteinase _ (MMP) -1 and MMP-2 by fibroblasts from normal gingiva and hereditary_gingival_fibromatosis .
IL-6 levels were much higher in patients with alpha-hemolytic streptococcus SS than in controls with uncomplicated bacteremia due to gram-positive organisms but were comparable with those in controls with bacteremia due to gram-negative organisms .
Albumin secretion rates , which were reduced by a factor of 2 in response to either heat_shock or IL-6 stimulation alone , were down-regulated by a factor of 4 when IL-6 was administered simultaneously with a continuous 40 degrees C heat_shock .
The effects of heat_shock on acute phase protein synthesis can be modified by preincubation with IL-6 , whereas addition of this ligand after heat treatment has no effect on the suppressive effect of heat on the APPR .
IL-6 and IL-8 concentrations were elevated immediately after ESWT and remained significantly elevated for four hours post-ESWT ($p < 0.001$) .
The analyses showed higher levels of IL6 in sera from patients with STSS than in sera from patients with bacteremia without shock .
Unlike heat_shock , activation of HSF by either hypo - or hyper-osmotic stress did not lead to an accumulation of heat-shock_protein_70 (HSP70) mRNA in HeLa cells .
NMNAT is transcriptionally regulated during various stress conditions including heat_shock and hypoxia through heat_shock factor (HSF) and hypoxia-inducible factor 1a in vivo .
Similar inhibition by Rac1N17 of HSF activation in response to heat_shock was observed .
The present study shows that under conditions of spermidine depletion caused by alpha-difluoromethylornithine , the DNA binding capacity of the transcription factor HSF induced by heat_shock undergoes a severe and prompt deactivation .
In the present study , we investigated whether the binding activity of heat_shock_transcription_factor (HSF) to the heat_shock element (HSE) of the hsp72 gene promoter increased after UV irradiation of human glioblastoma A-172 cells .
Heat_shock induces c-Jun_N-terminal_kinase (JNK) activation as well as heat_shock protein (HSP) expression through activation of the heat_shock_factor (HSF) , but its signal pathway is not clearly understood .
Interleukin-6 is the strongest predictor of 30-day mortality in patients with cardiogenic_shock due to myocardial_infarction .
CONCLUSIONS : Interleukin-6 concentrations are an independent predictor of 30-day mortality in patients with acute_myocardial_infarction complicated by cardiogenic_shock .
CONCLUSION : Patients with cardiogenic_shock who did not survive up to 28 days showed a decline in activated protein C levels during the course of the disease , which was inversely correlated with interleukin-6 .
METHODS : We measured serum activated protein C and interleukin-6 levels in 43 patients with cardiogenic_shock following acute myocardial_infarction and in 15 control patients with uncomplicated myocardial_infarction at days 0-5 and 7 after the onset of shock/myocardial _ infarction .
Plasma concentrations of interleukin-6 , organ failure , vasopressor support , and successful coronary revascularization in predicting 30-day mortality of patients with cardiogenic_shock complicating acute_myocardial_infarction .
Multiple_organ_failure in patients with cardiogenic_shock is associated with high plasma levels of interleukin-6 .
MEASUREMENTS AND MAIN RESULTS : Patients with CS had higher IL-6 levels than noncritically ill controls ($p < .001$) but lower levels than patients with septic_shock ($p = .003$) .

We studied IL-6 plasma levels in patients with CS with respect to organ failure .
Although organ failure is also a common complication of cardiogenic_shock (CS) , IL-6 levels have been reported to be lower in patients with CS than in patients with septic_shock .
The maximum level of IL6 was detected between 12 to 24 hours after the onset of MI among patients with cardiogenic_shock .
OBJECTIVE : Interleukin-6 (IL-6) , which is increased in patients who are suffering from septic_shock , is an important mediator of the inflammatory response .
[Interleukin-6 in hematological_diseases with septic_shock] .
Levels of soluble CD14 , interleukin-6 (IL-6) , IL-6_receptor (IL-6R) , and C-reactive_protein (CRP) have been measured in plasma from 26 children with septic_shock (nine of whom had disseminated intravascular_coagulation) and from ten control children .
The prognostic value of ADAMTS13 (a disintegrin and metalloprotease with thrombospondin type 1 repeats , member 13) deficiency in septic_shock patients involves interleukin-6 and is not dependent on disseminated_intravascular_coagulation .
We investigated here the effects of endotoxin removal by polymyxin B-immobilized polystyrene fiber (PMX-F) treatment on circulating levels of HMGB1 , soluble RAGE (sRAGE) , and interleukin-6 (IL-6) in septic_shock patients .
PURPOSE AND PATIENTS : We measured the serum concentrations of interleukin-6 (IL-6) in 70 patients with established septic_shock caused predominantly by gram-negative bacteria .
The interleukin-6 (IL6) -174_G / C promoter genotype is associated with the presence of septic_shock and the ex vivo secretion of IL6 .
Our results demonstrate that VIP and PACAP decrease lipopolysaccharide (LPS) - induced interleukin-6 (IL-6) production , neutrophil infiltration and intercellular_adhesion_molecule-1 (ICAM-1) , vascular_cell_adhesion_molecule-1 (VCAM-1) , and fibrinogen expression through PAC1 receptor , providing an advantage to design more specific drugs complementing standard intensive care therapy in septic_shock .
By facilitating the activity of interleukin-6 , it is likely that alterations in the levels of soluble interleukin-6_receptor in septic_shock could affect the severity of disease .
Changes in the interleukin-6 / soluble interleukin-6_receptor axis in meningococcal septic_shock .
In serial measurements , interleukin-6 peak values constantly preceded those of thrombopoietin , whereas peaks in thrombopoietin levels coincided with clinical episodes of septic_shock .
In the last years pro-calcitonin , interleukin-6 , C-reactive_protein , and nitric_oxide from endothelial and muscularis cells have been evaluated as prognostic factors in the septic_shock .
The geometric mean interleukin-6 level in these culture-positive patients was 407 pg/mL (95 % confidence interval , 108 to 1,545) ; all three children with levels of more than 300 pg/mL developed septic_shock , and one died .
CONCLUSION : High levels of interleukin-6 occur in children with septic_shock , and the presence of interleukin-6 in serum is predictive for the isolation of bacteria from blood and/or spinal fluid .
Plasma atrial natriuretic peptide and brain_natriuretic_peptide are increased in septic_shock : impact of interleukin-6 and sepsis-associated left_ventricular_dysfunction .
The only parameter that showed correlation with disease severity was the increase in interleukin-6 in final phase of sepsis , which corresponds to septic_shock .
OBJECTIVE : In patients with septic_shock , multiple_organ_failure (MOF) is associated with high levels of interleukin-6 (IL-6) .
Similar to interleukin_6 , abnormally high serum erythropoietin levels appear to be a negative prognostic indicator in patients suffering from septic_shock .

Plasma levels of cytokines , including interleukin_6 (IL-6) , IL-8 , IL-10 , and high-mobility_group_box_1 , were elevated in patients with septic_shock compared with healthy controls , but cytokine levels were not altered by PMX-DHP .
DESIGN : PCT , C-reactive_protein (CRP) , interleukin_6 (IL-6) dosages were sampled in four groups of patients : septic_shock patients (SS group) , shock_without_infection (NSS group) , patients with systemic inflammatory response syndrome related to a proven bacterial_infection (infect .
Furthermore , concentrations in serum of interleukin_6 strongly predicted degree of myocardial_dysfunction and severity of disease in children with meningococcal septic_shock .
Role of interleukin_6 in myocardial_dysfunction of meningococcal septic_shock .
VPS13D Gene Variant Is Associated with Altered IL-6 Production and Mortality in Septic_Shock .
In postoperative septic_shock monocytes may be almost areactive towards natural stimuli like bacteria and endotoxin , since IL-6 and TNFalpha production decreased to very low amounts .
Effect of L-NAME , an inhibitor of nitric_oxide synthesis , on plasma levels of IL-6 , IL-8 , TNF_alpha and nitrite/nitrate in human septic_shock .
We have confirmed the finding of IL-6 as a sensitive and reliable inflammatory marker in septic_shock .
Levels of soluble CD14 , interleukin-6 (IL-6) , IL-6_receptor (IL-6R) , and C-reactive_protein (CRP) have been measured in plasma from 26 children with septic_shock (nine of whom had disseminated intravascular_coagulation) and from ten control children .
CSF IL-6 after TBI is similar to serum IL-6 levels previously reported in children with septic_shock .
CSF IL-6 after TBI is similar to serum IL-6 levels previously reported in children with septic_shock .
OBJECTIVE : To compare the patterns of evolution of two proinflammatory cytokines (tumor_necrosis_factor _ [TNF] - alpha and interleukin-6 [IL-6]) in two major clinical entities associated with systemic inflammatory response : septic_shock and multiple trauma (with and without hemorrhagic_shock) .
Higher IL-6 concentrations were maintained throughout the study period in septic_shock patients than in trauma patients ($p < .001$) .
In survivors , at study entry , IL-6 concentrations were much higher in septic_shock patients than in trauma patients (3947 + / - 1410 vs. 247 + / - 41 pg/mL ; $p < .001$) .
During the whole study period , much higher concentrations of IL-6 were detected in septic_shock patients than in trauma patients ($p < .0001$) .
At study entry , IL-6 concentrations were significantly higher in nonsurvivor septic_shock patients than in nonsurvivor trauma patients (15,627 + / - 4336 vs. 317 + / - 124 pg/mL ; $p < .0001$) .
Together , the present results strongly support the hypothesis that the decrease in plasma CBG concentrations is associated with the increase in IL-6 and glucocorticoid levels reported in patients with septic_shock and burn_injury .
Our results showed that in patients with septic_shock , of the inflammatory cytokines , only IL-6 showed significantly higher plasma levels in the nonsurvivor group .
Starting within 2 h of fever onset , IL-6 levels rose significantly over baseline in both groups to markedly higher levels in patients with evolving septic_shock (medians : 372 vs. 3671 pg/mL ; $P < .001$) .
In addition to IL-6 , TNF-alpha was proved to be the mediator that orchestrates the hemodynamic and tissue_injury in septic_shock .
High IL-6 serum levels are associated with septic_shock and mortality in septic patients with severe leukopenia due to hematological_malignancies .
In evolving septic_shock , IL-6 , IL-8 and TNF-alpha peaked within 48 h of fever onset at levels reported for non-leucocytopenic patients and distinctively higher than during uncomplicated febrile episodes ($P < 0.05$) .

Increased IL-1b , IL-6 , IL-8 , IL-10 , TNF-a and decreased C4 d , C5a and iC3b levels were associated with septic_shock , coma and mortality .
(4) The predictive power of IL-6 , IL-10 and PCT combination (AUC = 0.994) was superior to IL-10 alone (AUC = 0.810) in predicting septic_shock (Z = 10.211 , P < 0.01) .
CONCLUSIONS : In this patient population with predominantly septic_shock and multiple_organ_failure , hemoadsorption removed IL-6 but this did not lead to lower plasma IL-6-levels .
These data provide evidence for a role of IL-6 in the pathophysiology of septic_shock .
IL-6 on admission appeared to be of prognostic significance : levels were higher in septic patients who subsequently died than in those who survived (P = .0003) , in particular when only patients with septic_shock were considered (P less than .0001) .
The highest IL-6 levels were encountered in patients who suffered from septic_shock (P value of the difference between patients with and without shock less than .0001) .
The rise of IL-6 during HCO-CVVHD seems to be a marker of bad prognosis in septic_shock patients .
Median serum concentration of IL-6 was 1,000 times higher in patients with septic_shock (189 ng/ml) than in patients with bacteriaemia , meningitis , or combined septic_shock and meningitis .
OBJECTIVE : To study the effects of Ringer 's sodium_pyruvate solution on tumor_necrosis_factor-a (TNF-a) and interleukin-6 (IL-6) upon septic_shock .
OBJECTIVE : Interleukin-6 (IL-6) , which is increased in patients who are suffering from septic_shock , is an important mediator of the inflammatory response .
Patients with septic_shock showed increased levels of IL-6 , IL-8 , MCP-1 , MIP-1b , IFN-y , GM-CSF and IL-10 compared to healthy controls .
IL-6 and IL-10 levels were positively associated with septic_shock and mortality rates .
RESULTS : Systolic_and_diastolic_blood_pressures were significantly lower , and endotoxin , IL-6 , HMGB1 , and sRAGE levels were higher in septic_shock patients compared with healthy volunteers .
Our serial observations suggest that , in human septic_shock , peripheral vasodilation is most strongly and independently , of all inflammatory factors , associated with IL-6 release , whereas complement activation partly offsets the myocardial_depression of the syndrome .
Plasma levels of cytokines , including interleukin_6 (IL-6) , IL-8 , IL-10 , and high-mobility_group_box_1 , were elevated in patients with septic_shock compared with healthy controls , but cytokine levels were not altered by PMX-DHP .
While the production of tumor_necrosis_factor (TNF) and interleukin-6 (IL-6) in septic_shock and other inflammatory states is well established , the role of interleukin-8 (IL-8) , a recently described neutrophil chemoattractant and activator , has yet to be fully elucidated .
It has also been suggested that IL-6 is involved in the pathogenesis of septic_shock .
Three important cytokines associated with septic_shock are tumour_necrosis_factor / cachectin (TNF) , interleukin_1 _ (IL-1) and interleukin_6 (IL-6) .
Circulating levels of advanced glycation end products (AGE) and interleukin-6 (IL-6) are independent determinants of serum asymmetric_dimethylarginine (ADMA) levels in patients with septic_shock .
IL-6 was comparatively more associated with septic_shock and IL-10 was comparatively more associated with mortality .
CONCLUSIONS : These results indicate that circulating levels of IL-6 are detectable in a majority of patients with gram-negative septic_shock .
DESIGN : PCT , C-reactive_protein (CRP) , interleukin_6 (IL-6) dosages were sampled in four groups of patients : septic_shock patients (SS group) , shock_without_infection (NSS group) , patients with systemic inflammatory response syndrome related to a proven bacterial_infection (infect .
Forty-three patients with septic_shock were assessed by monitoring of blood IL-6 level with a rapid assay system and immediate initiation of critical care including PMMA-CHDF for cytokine removal .

Among the patients who developed septic_shock , a high IL-6 blood level and a low LF were observed in both the survivor and nonsurvivor groups on the day of admission .
IL-6 is frequently released into serum during septic_shock , and its levels are associated with the severity of the shock .
RESULTS : Concentrations of IL-1_beta , IL-6 , IL-7 , IL-8 , IL-10 , IL-13 , interferon-gamma , MCP-1_and_tumour_necrosis_factor-alpha were significantly higher in septic_shock patients than in those with severe sepsis .
Our results demonstrate that VIP and PACAP decrease lipopolysaccharide (LPS) - induced interleukin-6 (IL-6) production , neutrophil infiltration and intercellular_adhesion_molecule-1 (ICAM-1) , vascular_cell_adhesion_molecule-1 (VCAM-1) , and fibrinogen expression through PAC1 receptor , providing an advantage to design more specific drugs complementing standard intensive care therapy in septic_shock .
Elevated systemic levels of proinflammatory cytokines (IL-1beta , IL-6 , IL-8 and IL-10) at the time of diagnosis of hospital-acquired_pneumonia appear to be indicative of subsequent progression to septic_shock .
Results from a phase II clinical trial on 122 patients in Germany did not show prolonged survival in patients with septic_shock but did indicate lowered levels of IL-6 .
MATERIALS AND METHODS : We measured serum levels of IL-6 , IL-8 , sELAM-1 and sICAM-1 in 40 intensive care unit patients who developed septic_shock .
We investigated the predictive value of the mediators IL-6 , IL-8 , sELAM-1 and sICAM-1 and their time course in intensive care unit patients who developed septic_shock with respect to outcome .
A significantly larger proportion of children with high IL-1Ra : TNF-alpha and IL-1Ra : IL-6 ratios developed severe disease with septic_shock than those with a low ratios (95.2 % vs. 4.8 % ; 76.2 % vs. 23.8 %) .
MEASUREMENTS AND RESULTS : Median IL-1Ra : TNF and IL-1Ra : IL-6 ratios were significantly higher in severe disease with septic_shock than in severe disease without septic_shock and in non severe disease (IL-1Ra : TNF 263 vs. 185 vs. 108 ; IL-1Ra : IL-6 139 vs. 23 vs. 17) .
MEASUREMENTS AND RESULTS : Median IL-1Ra : TNF and IL-1Ra : IL-6 ratios were significantly higher in severe disease with septic_shock than in severe disease without septic_shock and in non severe disease (IL-1Ra : TNF 263 vs. 185 vs. 108 ; IL-1Ra : IL-6 139 vs. 23 vs. 17) .
There was a direct correlation between IL-6 peak serum level and TNF_alpha peak serum level during septic_shock and between IL-6 serum level and temperature or C-reactive_protein serum level .
Interleukin_1_beta (IL-1_beta) , tumor_necrosis_factor (TNF_alpha) , and interleukin_6 (IL-6) were measured each day and every 1 or 2 hours when septic_shock occurred .
These data revealed the importance in the level of IL-6 , rather than endotoxin and CRP , in managing the patients with septic_shock .
IL-6 was higher in patients who developed septic_shock , compared with patients who had only sepsis .
We defined two types of septic_shock from these data , i.e. , endotoxin + TNF-alpha + IL-2 shock and IL-beta + IL-6 shock .
Circulating levels of IL-6 and sPLA (2) were higher in patients developing septic_shock and in nonsurvivors , particularly in Group 1 .
No significant differences in IL-6 levels were found when comparing CS patients with MOF at the time of blood sampling with patients with septic_shock .
OBJECTIVE : In patients with septic_shock , multiple_organ_failure (MOF) is associated with high levels of interleukin-6 (IL-6) .
CONCLUSIONS : Once MOF is present , patients with CS exhibit similarly high IL-6 levels as patients with septic_shock .

On admission , tumor_necrosis_factor-alpha , IL-6 , procalcitonin , and lipopolysaccharide-binding protein levels were higher in patients with septic_shock than in patients with multitrauma .
In blood samples of patients in prolonged septic_shock , epinephrine did not modulate cytokine levels of IL-6 and IL-10 , and decreased TNFalpha only by 36.4 % (P < 0.0001) .
Plasma AM and IL-8 levels correlated positively with Acute Physiology and Chronic Health Evaluation (APACHE) II score , peak multiple_organ_failure (MOF) score during the first month and prognosis in patients with septic_shock , as did plasma IL-6 levels in patients with traumatic_shock .
IL_6 is certainly involved in the pathophysiology of septic_shock but further studies are required to determine whether or not it is directly involved in the mediation of late and lethal complications of septic_shock .
The initial levels of IL6 were comparatively higher in patients with septic_shock and non-survivors , and increased at 48 hr of admission in patients with sepsis , septic_shock and non-survivors .
RESULTS : Concentrations of IL-1_beta , IL-6 , IL-7 , IL-8 , IL-10 , IL-13 , interferon-gamma , MCP-1_and_tumour_necrosis_factor-alpha were significantly higher in septic_shock patients than in those with severe sepsis .
Insulin response during hypovolemic_shock .
Insulin secretion in cardiogenic_shock .
Insulin secretion following myocardial_infarction with particular respect to the pathogenesis of cardiogenic_shock .
[Lack of incretion of insulin in the blood in reversible cardiogenic_shock] .
The Effect of Nutrition on Early Stress-Induced Hyperglycemia , Serum Insulin Levels , and Exogenous Insulin Administration in Critically Ill Patients with Septic_Shock : A Prospective Observational Study .
CONCLUSIONS : In patients with septic_shock marked reduced serum insulin levels can be observed during the first 36hours after ICU admission that have to be compensated by exogenous insulin administration , a phenomenon gradually improving after 36hours .
We show here , using a conditional knockout of Irf6 in lysosymeM expressing cells , that Irf6 is required for resistance to LPS-induced endotoxic_shock .
We show here , using a conditional knockout of Irf6 in lysosymeM expressing cells , that Irf6 is required for resistance to LPS-induced endotoxic_shock .
Collectively , our findings suggest a role for Irf6 in the resistance to endotoxic_shock due to NFk-B-mediated alteration of cytokine production .
Patients with septic_shock and sepsis had significantly increased neutrophil CD11b expression compared with normal subjects .
CONCLUSIONS : The results suggest that blockade of LBP at inflammation sites might attenuate LPS-induced circulatory shock .
Serum LBP at study entry was statistically significantly lower in patients with SIRS than in those with septic_shock (P < 0.014) ; no statistically significant difference existed between patients with SIRS and those with sepsis (P = 0.61) .
Role of Urinary Neutrophil_Gelatinase-associated_Lipocalin as a Biomarker of Acute_Kidney_Injury in Patients with Circulatory_Shock .
Plasma NGAL was significantly increased within 7 days before AKI development in total patients (P < .001) and septic_shock patients (P < .001) but not significantly increased in patients without septic_shock (P = .167) .
The area under the ROC curve showed that plasma NGAL as a predictor of death in septic_shock was significant .
ELISA detection of circulating levels of LIF , OSM , and CNTF in septic_shock .
Leukemia_inhibitory_factor (LIF) has recently been associated with septic_shock in humans .

Leukemia_inhibitory_factor protects against experimental lethal Escherichia_coli septic_shock in mice .
Leukemia_inhibitory_factor levels are elevated in septic_shock and various inflammatory body fluids .
Considering LILRB2 inhibitory properties , we can hypothesize that LILRB2 may participate in the altered immune response after septic_shock .
Septic_shock was associated with the LTalpha +250 : TNFalpha-308 A : G haplotype but not the A : A haplotype , suggesting that LTalpha +250 is a marker , rather than a causative polymorphism .
Septic_shock was associated with the LTalpha +250 : TNFalpha-308 A : G haplotype but not the A : A haplotype , suggesting that LTalpha +250 is a marker , rather than a causative polymorphism .
In the absence of septic_shock , there was a significant trend to greater T1RF in patients with LTalpha +250 GG (TNFalpha hyposecretor) genotype ($p = 0.03$) .
UNASSIGNED : Excessive activation of Toll-like_receptor_4 (TLR4) / MD-2 by lipopolysaccharide (LPS) causes septic_shock .
The data presented in this report strongly suggest that LZM-LPS complex formation completely abrogates tumor_necrosis factor production and the mortality caused by LPS and that LZM may be useful for the treatment of endotoxin shock .
In this study , we examined the effect of LZM on the LPS-triggered septic_shock model induced by carrageenan treatment and assessed by tumor_necrosis factor production .
Osmotic_shock also induces transient ERK activation in ECs .
Heat_shock up-regulates TLR9 expression in human B cells through activation of ERK and NF-kappaB signal pathways .
Taken together , our results suggest that ERK and JNK play an important role in the induction of MMP-1 and MMP-3 by heat_shock and that the heat_shock-induced expression of MMP-1 and MMP-3 is mediated via an IL-6-dependent autocrine mechanism .
Inhibiting ERK MAPK activation during heat_shock with PD98059 enhanced losses in cell viability .
When MII oocytes were heated , only the phosphorylated p38 levels relative to the total p38 levels decreased ($P < 0.01$) after HS , but no clear relationship with HS treatments was observed in the ERK , JNK and p90 (rsk) expressions of matured oocytes .
From the PPI network , the top 10 hub genes , which are all upregulated DEGs in the septic_shock children , were identified as GAPDH , TNF , EGF , MAPK3 , IL-10 , TLR4 , MAPK14 , IL-1b , PIK3CB , and TLR2 .
The proteasome mediated step (s) in apoptosis is located upstream of mitochondrial changes and caspase activation , and can involve in different systems Bcl-2 , Jun_N-terminal_kinase , heat_shock proteins , Myc , p53 , polyamines and other factors .
Pretreatment with 1,25 (OH) (2) D (3) inhibited the activation of JNK by all stresses and the activation of p38 by heat_shock , AG1478 and tumor_necrosis_factor_alpha .
Since Rac1V12 was able to activate JNK , it is suggested that heat_shock may activate JNK via Rac1 .
In addition , Rac1N17 , a dominant negative mutant of Rac1 , significantly inhibited JNK activation by heat_shock .
Therefore , we tested whether heat_shock activates JNK via inhibition of JNK dephosphorylation .
Heat_shock induces c-Jun_N-terminal_kinase (JNK) activation as well as heat_shock protein (HSP) expression through activation of the heat_shock_factor (HSF) , but its signal pathway is not clearly understood .
The lowest MBL levels were detected in those infants with septic_shock , particularly those who died ($p < 0.05$) .

Plasma concentration of MIF correlated with disease severity , the presence of shock and with the cytokines interleukin _ (IL) _ 1beta , IL-10 , IL-12 , and vascular_endothelial_growth_factor , but not with TNF-alpha .
MIF has been found to be a mediator of several diseases including gram-negative septic_shock and delayed-type_hypersensitivity reactions .
INTRODUCTION : Macrophage_migration_inhibitory_factor (MIF) plays an essential pathophysiological role in septic_shock , but its role_in_central_nervous_system_infection (CNS) remains to be defined .
Macrophage_migration_inhibitory_factor (MIF) in meningococcal septic_shock and experimental human endotoxemia .
The median plasma concentrations of MIF and IL-6 were significantly higher in patients with septic_shock and in patients with sepsis than in healthy controls .
The levels of MIF and IL-6 were measured in 25 patients with septic_shock , 17 patients with sepsis , and 11 healthy volunteers .
Our data suggest that during immune-mediated inflammation (such as septic_shock) MIF is an important neuroendocrine mediator : a contraregulator of the immunosuppressive effects of glucocorticoids .
Emerging studies indicate that MIF plays a pivotal role not only in endotoxic_shock but also in the host response to a variety of acute_and_chronic_infections .
MIF plays a pivotal role in the host response to endotoxic_shock and appears to serve as a pituitary `` stress '' hormone that regulates systemic inflammatory responses .
MIF is a pivotal mediator in endotoxic_shock and may serve as a pituitary `` stress '' hormone that regulates systemic inflammatory responses .
MIF , a previously unrecognized pituitary hormone and macrophage cytokine , is a pivotal mediator in endotoxic_shock .
The administration of MIF increases lethality during endotoxemia , whereas neutralization of this cytokine prevents endotoxic_shock and death associated with bacterial_infection .
INTRODUCTION : Macrophage_migration_inhibitory_factor (MIF) plays an essential pathophysiological role in septic_shock , but its role_in_central_nervous_system_infection (CNS) remains to be defined .
OBJECTIVE : Macrophage_migration_inhibitory_factor (MIF) has emerged as an important mediator of septic_shock .
Macrophage_migration_inhibitory_factor (MIF) is a unique cytokine and critical mediator of host defenses with a role in septic_shock and chronic inflammatory and autoimmune_diseases .
BACKGROUND : The human (Homo_sapiens) chemokine-like protein macrophage_migration_inhibitory_factor (HsMIF) is a pivotal mediator of inflammatory , infectious and immune_diseases including septic_shock , colitis , malaria , rheumatoid_arthritis , and atherosclerosis , as well as tumorigenesis .
Is macrophage_migration_inhibitory_factor (MIF) the `` control point '' of vascular hypo-responsiveness in septic_shock ?
We report that APC inhibits in vitro the release of tumor_necrosis factor (TNF) and macrophage_migration_inhibitory_factor (MIF) , two known cytokine mediators of bacterial_septic_shock , from lipopolysaccharide (LPS) - stimulated human monocytes .
In patients with septic_shock (n = 32) , multitrauma (n = 8) , and hospitalized matched controls (n = 41) , we serially measured serum macrophage_inhibitory_factor (MIF) , cortisol , plasma ACTH , tumor_necrosis_factor-alpha , and interleukin-6 (IL-6) immunoreactivity during 14 days or until discharge/death .

Furthermore , miRNA-155 levels were significantly higher in the T cells from patients with septic_shock , as compared with those from healthy volunteers ($P < 0.05$) .
We investigated MMP-9 activity in myocardial_infarction-induced cardiogenic_shock with regard to RAGE/sRAGE regulation .
Plasma MMP-9 concentrations and monocyte MMP-9 mRNA levels were significantly higher in the 10 nonsurviving patients with septic_shock than in 10 surviving patients and 25 normal controls .
In human studies , plasma levels of MMP-9 were significantly increased in patients with septic_shock as compared with healthy controls , although MMP-9 levels did not correlate with organ_injury score .
The mammalian_target_of_rapamycin (mTOR) is a critical mediator of the phosphoinositide 3-kinase/protein _ kinase_B / mTOR signaling pathway , and mTOR activity is induced following heat_shock .
The elevated level of NO3 - during the initial several days in septic_shock will mean that NO is acting to prevent platelet_aggregation and to keep blood flow by dilating the arteries during septic_shock .
Heat_shock induces the cellular antioxidant defenses peroxiredoxin , glutathione and glucose_6-phosphate_dehydrogenase through Nrf2 .
This study aims to understand the role of the Nrf2 antioxidant pathway in acquisition of mild thermotolerance at 40 C , and secondly , whether the Nrf2 pathway could be involved in the protective effect of thermotolerance against heat-shock (42 C) - induced apoptosis .
Moreover , Nrf2 contributes to the protective effect of thermotolerance against heat-shock (42 C) - induced apoptosis , because Nrf2 activation by oltipraz enhanced thermotolerance , whereas Nrf2 knockdown partly reversed thermotolerance .
Moreover , Nrf2 contributes to the protective effect of thermotolerance against heat-shock (42 C) - induced apoptosis , because Nrf2 activation by oltipraz enhanced thermotolerance , whereas Nrf2 knockdown partly reversed thermotolerance .
Moreover , Nrf2 contributes to the protective effect of thermotolerance against heat-shock (42 C) - induced apoptosis , because Nrf2 activation by oltipraz enhanced thermotolerance , whereas Nrf2 knockdown partly reversed thermotolerance .
It was previously shown that heat_shock , as well as NSAIDs , inhibits IR-induced activation of NF-kappaB and that NF-kappaB protects against IR-induced cytotoxicity .
Autophagy activation by NFkappaB is essential for cell survival after heat_shock .
By contrast , the values of P50 in vivo were significantly increased in patients with cardiogenic_shock .
Heat_shock induces IkappaB-alpha and prevents stress-induced endothelial cell apoptosis .
CONCLUSIONS : These findings indicate that the HSR inhibits cytokine-induced NOS2 expression and NO synthesis in AKN-1 cells by preventing NOS2 promoter activation .
AIMS : Previous studies suggested haemodynamic benefits and , possibly , mortality reduction with the use of nitric_oxide_synthase (NOS) inhibition in patients with acute_myocardial_infarction (AMI) complicated by cardiogenic_shock (CS) .
There is limited evidence regarding the degree of iNOS induction in human cells or tissues with septic_shock .
On admission , the septic_shock patients demonstrated significantly higher levels of PhLA (2.3 vs. 0.8 mol/L) , p-HPhAA (4.6 vs. 1.4 mol/L) , p-HPhLA (7.4 vs. 2.6 mol/L) , HVA , lactate , and significantly lower levels of iNOS .
PGP inhibited the production of NO and ROS and expression of iNOS , COX-2 , TNF-a and IL-1b , which are involved in the pathogenesis of many inflammation-associated human diseases , including septic_shock , hemorrhagic_shock and rheumatoid_arthritis .
CONCLUSIONS : In patients with septic_shock , neuronal apoptosis is rather associated with iNOS expression and microglial apoptosis with hyperglycaemia , possibly because GLUT5 is not downregulated .

Inducible_nitric_oxide_synthase (iNOS) is involved in many physiological and pathophysiological processes , including septic_shock and acute_kidney_failure .
Apoptosis of neurons in cardiovascular autonomic centres triggered by inducible_nitric_oxide_synthase after death from septic_shock .
UNASSIGNED : The aim of this study was to determine whether systemic_inflammatory_response_syndrome (SIRS) in burn patients is mediated by the brain_natriuretic_peptide (BNP) / natriuretic peptide A receptor (NPRA) - induced heat_shock factor 1 (HSF-1) signaling pathway .
METHODS : The plasma BNP and invasive hemodynamic parameters data [central venous pressure (CVP) , pulmonary arterial pressure (PAP) , pulmonary capillary wedge pressure (PCWP) , and cardiac output (CO)] were collected from 21 noncardiac_shock patients received Swan-Ganz catheterization throughout a continuous surveillance for 3 days .
But in noncardiac_shock cases , the increased BNP did not correlate with heart function , therefore it could not replace the Swan-Ganz catheter data to guide the treatment in these patients .
However , only BNP plasma levels were reduced by MCS , whereas the concentrations of remodeling biomarkers remained elevated or even increased further 30 days after MCS .
LV systolic_dysfunction is associated with a higher severity of illness , use of vasoactives , and BNP , whereas RV systolic_dysfunction is associated with cold_shock .
CONCLUSION : In critically_ill patients with acute_circulatory_failure , BNP does not accurately predict fluid responsiveness .
B-type_natriuretic_peptide and its molecular precursor in myocardial_infarction complicated by cardiogenic_shock .
CONCLUSIONS : While routine parameters did not predict the clinical course , elevated BNP and E-selectin independently predicted cardiogenic_shock on admission and 1 day before its occurrence .
Recently , elevated brain_natriuretic_peptide (BNP) levels have been observed in patients with septic_shock .
Plasma BNP was higher in SS than in cardiogenic_shock patients (1367 + / - 1438 vs. 750 + / - 346 respectively ; p = 0.027) .
Plasma BNP is very high in septic-shock patients (> 1000 pg/ml) , which is suggested to relate to both myocardial stretch and to an alteration in one of the BNP clearance pathways .
RESULTS : Patients with septic_shock had significantly higher BNP levels on admission compared with the other 2 groups (P < .05) .
Plasma levels of BNP and ANP were markedly elevated in patients with septic_shock / severe sepsis compared with controls (BNP , 7 + / - 0.3 pg mL ; ANP , 13 + / - 1 pg mL) .
However , growing evidence supports the hypothesis that BNP could be an early predictor of mortality in septic_shock .
Detection of C-type_natriuretic_peptide in human circulation and marked increase of plasma CNP level in septic_shock patients .
Glucocorticoid_receptor (GR) recycles between an inactive form complexed with heat_shock proteins (hsps) and localized to the cytoplasm and a free liganded form that regulates specific gene transcription in the nucleus .
Immunophilins , Heat_Shock Proteins , and Glucocorticoid_Receptor Actions in Vivo The glucocorticoid_receptor is a member of the steroid/thyroid receptor family and , as such , acts as a ligand-inducible enhancer of specific gene transcription .
Heat_shock protein is tightly associated with the recombinant human glucocorticoid_receptor : glucocorticoid response element complex .
Heat_shock and other forms of stress increase glucocorticoid_receptor (GR) activity in cells , suggesting cross-talk between the heat_shock and GR signal pathways .

Geldanamycin , a heat_shock protein 90-binding benzoquinone_ansamycin , inhibits steroid-dependent translocation of the glucocorticoid_receptor from the cytoplasm to the nucleus .
The glucocorticoid_receptor (GR) is a ligand-regulated transcription factor whose ability to bind hormone is thought to be dependent on association with the 90-kDa heat_shock protein (hsp90) .
When isolated from cells grown under hormone-free conditions , the glucocorticoid_receptor (GR) is known to exist as a large heteroprotein complex that contains , among its multiple components , the stress protein hsp90 (heat_shock protein 90) .
Stable and specific binding of heat_shock protein 90 by geldanamycin disrupts glucocorticoid_receptor function in intact cells .
Previously , it has been found that glucocorticoid_receptor (GR) binding activity decreased rapidly during heat_shock response in HOS-8603 , a human osteosarcoma cell line .
The unliganded glucocorticoid_receptor is a multi-oligomer complex consisting of a ligand-binding protein with which two 90 kDa heat_shock proteins (hsp90s) are associated .
These effects were dose-dependent and appeared to be mediated via the glucocorticoid_receptor , because combined exposure to dexamethasone or RU_28362 plus RU_38486 completely restored synthesis and expression of the 72 kDa heat_shock protein .
In the presence of certain metals , regions of the hormone binding domain of the glucocorticoid_receptor (GR) are capable of binding the 90 kDa heat_shock protein (hsp90) .
The role of heat_shock proteins in regulating the function , folding , and trafficking of the glucocorticoid_receptor .
Differential roles of heat_shock protein 70 in the in vitro nuclear import of glucocorticoid_receptor and simian virus 40 large tumor antigen .
Effects of heat_shock on glucocorticoid_receptor .
Point mutations in the 90-kDa heat_shock protein binding region of the glucocorticoid_receptor affect the functional characteristics of the receptor .
Here we show that under conditions permitting minimal in vitro manipulation , the steroid-free glucocorticoid_receptor in crude cytosol associates with the hsp90 heat_shock protein (relative molecular mass Mr approximately equal to 90,000) to form a large 300K complex , rather than the 94K liganded receptor monomer .
The Mr approximately equal to 90,000 heat_shock protein , hsp90 , readily interacts with the glucocorticoid_receptor to form the 9 S , non-DNA-binding receptor complex .
Association of the dioxin receptor with the Mr 90,000 heat_shock protein : a structural kinship with the glucocorticoid_receptor .
The glucocorticoid_receptor is present in cytosol prepared from cell extracts of nonhormone-treated cells as a large nonactivated (i.e. non-DNA binding) 9 S heteromeric complex which contains the Mr approximately 90,000 heat_shock protein , hsp90 .
Such a locus that has received increasing attention is the gene encoding FK506_binding_protein_51 (FKBP5) , a heat_shock protein 90 cochaperone of the steroid receptor complex that among other functions regulates sensitivity of the glucocorticoid_receptor .
We have used three methods to measure the stoichiometry of the glucocorticoid_receptor and the 90-kDa heat_shock protein (hsp90) in L-cell glucocorticoid_receptor complexes that were purified by immunoadsorption to protein A-Sepharose with an anti-receptor monoclonal antibody , followed by a minimal washing procedure that permits retention of receptor-associated protein .
Compound A , a selective glucocorticoid_receptor modulator , enhances heat_shock protein Hsp70 gene promoter activation .
The glucocorticoid_receptor exists in the cytoplasm of hormone-untreated cells as a complex with the 90-kDa heat_shock protein (HSP90) .

Cytoplasmic 8 S glucocorticoid_receptor binds to actin filaments through the 90-kDa heat_shock protein moiety .
The 70-kDa heat_shock protein (Hsp70) is involved in providing the appropriate conformation of various nuclear hormone receptors , including the glucocorticoid_receptor (GR) .
In the case of the glucocorticoid_receptor (GR) , high-affinity ligand binding requires the interaction of the LBD with the heat_shock protein 90 (Hsp90) .
In 0.3 % albumin , corticosteroids reduced total ECM and collagen deposition , involving the glucocorticoid_receptor (GR) and downregulation of collagen , heat_shock protein 47 (Hsp47) , and Fli1 mRNA expression .
To further define the role of heat_shock factor 1 (HSF1) in the stress potentiation of glucocorticoid_receptor (GR) activity , we placed a constitutively active mutant of human HSF1 (hHSF1-E189) under the control of a doxycycline (DOX) - inducible vector .
We have previously shown that activation of glucocorticoid_receptor (GR) signaling in stressed cells will cause inhibition of the heat_shock response as mediated by heat_shock transcription factor 1 (HSF1) .
We have previously shown that activation of glucocorticoid_receptor (GR) signaling in stressed cells will cause inhibition of the heat_shock response as mediated by heat_shock transcription factor 1 (HSF1) .
Agonist-activated glucocorticoid_receptor inhibits binding of heat_shock factor 1 to the heat_shock_protein_70 promoter in vivo .
The inactive form of the glucocorticoid_receptor in the cytoplasm appears to be bound to heat_shock proteins of the 90K family (hsp90_alpha and hsp90_beta) .
Heat_shock and other forms of stress increase glucocorticoid_receptor (GR) activity in cells , suggesting cross-talk between the heat_shock and GR signal pathways .
Heat and chemical shock potentiation of glucocorticoid_receptor transactivation requires heat_shock factor (HSF) activity .
RECEPTOR ACTIVATION : When unstimulated , the glucocorticoid_receptor is inactivated by its integration within a multiple-protein complex associating heat_shock proteins , immunophilins and cyclophilins .
The heat_shock protein hsp70/hsc70 is a required component of a five-protein (hsp90 , hsp70 , Hop , hsp40 , and p23) minimal chaperone system reconstituted from reticulocyte lysate that forms glucocorticoid_receptor (GR) .
RESULTS : GR expression was higher in T lymphocytes from patients with septic_shock compared to healthy subjects ($p = 0.01$) .
However , dj1 and hsc70 apparently colocalized in the nucleoli after heat_shock .
Experimental and clinical studies suggest that PAF antagonists appear to be effective in cases of severe Gram-negative septic_shock .
PAF antagonists may have also therapeutical effects in septic_shock , in myocardial_ischemia and cardiac_rhythm_disturbances , in brain_damage following cerebral_ischemia and neurological_trauma , in gastric and intestinal damages or in some inflammatory reactions .
The findings suggest that endotoxin could interact with PAF to significantly augment possible hemorrhagic and/or thrombotic complications of septic_shock in humans .
A role for PAF in gastric_damage during septic_shock seems well established , and the clinical use of selective PAF antagonists in such situations seems appropriate .
This rapid release of PAF in response to endotoxin is consistent with a role for monocyte-derived PAF as a toxic mediator of the acute systemic changes observed in patients with endotoxin-related septic_shock .

This rapid release of PAF in response to endotoxin is consistent with a role for monocyte-derived PAF as a toxic mediator of the acute systemic changes observed in patients with endotoxin-related septic_shock .
The interrelationship of PAF and tumor_necrosis_factor (another key mediator of septic_shock) is also discussed .
The present review summarizes the biological effects of PAF and the effect of PAF antagonists in animal models of septic_shock .
Since the isolation and elucidation of the structure of platelet-activating_factor (PAF) in the late 1970 's , several preclinical studies have suggested that PAF is a key mediator of septic_shock induced in animals by either endotoxin or by Gram-negative bacteria .
Since the isolation and elucidation of the structure of platelet-activating_factor (PAF) in the late 1970 's , several preclinical studies have suggested that PAF is a key mediator of septic_shock induced in animals by either endotoxin or by Gram-negative bacteria .
CONCLUSIONS : These results demonstrate in humans during ARF associated with septic_shock the production of PAF , a mediator that has been previously implicated in the pathogenesis of experimental endotoxin-induced shock and renal_injury .
METHODS : The concentration of PAF and selected cytokines (TNF , IL-1 , IL-6 , IL-8) was evaluated in blood and urine of 12 patients with septic_shock and ARF for 4 consecutive days .
The present study was designed to determine whether the expression of PD-1 and PD-L1 is upregulated in septic_shock patients and to explore the role of this pathway in sepsis-induced immunosuppression .
Double immunofluorescence of hBD3 together with CD68 , CD31 , heat_shock protein 47 (HSP47) and mast_cell_tryptase (MCT) staining was done .
Prevention of PI3K by LY294002 blocked heat_shock / GD-induced apoptosis without reversing the cell death mode to necrosis , while inhibition of MEK1/2 by U0126 reversed heat_shock / GD-induced apoptosis to necrosis , indicating a different role (s) of PI3K and ERK1/2 in heat_shock / GD-induced cell_death_mode_determination .
Prevention of PI3K by LY294002 blocked heat_shock / GD-induced apoptosis without reversing the cell death mode to necrosis , while inhibition of MEK1/2 by U0126 reversed heat_shock / GD-induced apoptosis to necrosis , indicating a different role (s) of PI3K and ERK1/2 in heat_shock / GD-induced cell_death_mode_determination .
On the other hand , osmotic_shock activates a phospholipase_A2 leading to release of platelet activating factor , which in turn activates a sphingomyelinase and thus stimulates the formation of ceramide .
The structural , functional , regulatory and biologic characteristics of PLA2 are reviewed in relation to septic_shock and its complications and areas of controversy are highlighted .
The toxicity of phospholipase_A2 (PLA2) has been suggested to be involved in the pathology of a number of severe diseases including septic_shock and acute pancreatitis .
BACKGROUND : Because hypotension and pulmonary_injury have been associated with elevated PLA2 activity in septic_shock and PLA2 levels are reduced with the administration of glucocorticoids , the PLA2 response to endotoxin was investigated in volunteers pretreated with and without hydrocortisone .
BACKGROUND : Because hypotension and pulmonary_injury have been associated with elevated PLA2 activity in septic_shock and PLA2 levels are reduced with the administration of glucocorticoids , the PLA2 response to endotoxin was investigated in volunteers pretreated with and without hydrocortisone .
OBJECTIVE : To study blood and bronchoalveolar lavage (BAL) fluid levels of platelet activating factor (PAF-acether) and phospholipase_A2 (PLA2) in patients with septic_shock or following severe trauma .

PLA2 levels in 100 % of patients with septic_shock were elevated (as high as 43-fold over control levels) , and mean plasma PLA2 activity was increased 16-fold over control levels .
In an attempt to detect similar mechanisms in clinical gram-negative septic_shock , plasma samples from 34 patients with hypotension and septicemia were collected and assayed for PLA2 activity .
We tested a number of therapeutic agents for their ability to inhibit PLA2 from human septic_shock serum .
Elevated levels of circulating phospholipase_A2 (PLA2) correlate with the severity of circulatory_collapse and pulmonary_dysfunction in gram-negative septic_shock .
Since endogenous serum PLA2 levels correlate directly with the magnitude of hypotension in both experimental endotoxic_shock and clinical septic_shock , and since parenteral administration of purified exogenous PLA2 reproduces hypotension in experimental models , we conclude that high levels of intravascular PLA2 may contribute similarly to the circulatory collapse in septic_shock in man .
Since endogenous serum PLA2 levels correlate directly with the magnitude of hypotension in both experimental endotoxic_shock and clinical septic_shock , and since parenteral administration of purified exogenous PLA2 reproduces hypotension in experimental models , we conclude that high levels of intravascular PLA2 may contribute similarly to the circulatory collapse in septic_shock in man .
In pancreatitis , PLA2 levels paralleled fluctuations of serum amylase and lipase , whereas in septic_shock without pancreatic_involvement , PLA2 changes were discordant with changes in pancreatic enzymes .
In pancreatitis , PLA2 levels paralleled fluctuations of serum amylase and lipase , whereas in septic_shock without pancreatic_involvement , PLA2 changes were discordant with changes in pancreatic enzymes .
To assess the role of serum PLA2 in septic_shock in man , we determined serum PLA2 profiles in a prospective study in 12 patients with septic_shock .
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To assess the role of serum PLA2 in septic_shock in man , we determined serum PLA2 profiles in a prospective study in 12 patients with septic_shock .
In a prospective study of 10 episodes of septic_shock , serum PLA2 and cortisol levels correlated significantly in all survivors (p less than 0.0001) , whereas such a correlation was absent in all nonsurvivors (p less than 0.07) .
Because soluble PLA2 activity has been associated with circulatory collapse in hyperphospholipasemic_conditions , such as septic_shock and pancreatitis , we examined the relationship between circulating PLA2 activity and adrenocortical function .
Because soluble PLA2 activity has been associated with circulatory collapse in hyperphospholipasemic_conditions , such as septic_shock and pancreatitis , we examined the relationship between circulating PLA2 activity and adrenocortical function .
An autoregressive mathematical model was developed to describe the rate of PLA2 clearance during the recovery phase of septic_shock .
Circulating phospholipase_A2 (PLA2) has been recognized as a mediator of cardiovascular collapse in septic_shock .
Serum levels of phospholipase_A2 (PLA2) activity have been shown to be elevated in cases of septic_shock and rheumatoid_arthritis .
This article describes the novel finding of elevated PLA2 levels in cases of malaria , further strengthening the notion that mediators of the host response in cases of malaria are similar to those in cases of septic_shock .

Since endogenous serum PLA2 levels correlate directly with the magnitude of hypotension in both experimental endotoxic_shock and clinical septic_shock , and since parenteral administration of purified exogenous PLA2 reproduces hypotension in experimental models , we conclude that high levels of intravascular PLA2 may contribute similarly to the circulatory collapse in septic_shock in man .

Circulating phospholipase_A2 (PLA2) has been recognized as a mediator of circulatory collapse in experimental endotoxic_shock .

Phospholipase_A2 from plasma of patients with septic_shock is associated with high-density lipoproteins and C3 anaphylatoxin : some implications for its functional role .

Serum phospholipase_A2 enzyme activity and immunoreactivity in a prospective analysis of patients with septic_shock .

An increase in circulating levels of extracellular group II phospholipase_A2 (PLA2) has been detected in patients with septic_shock as well as in rats , rabbits and human volunteers following intravenous endotoxin administration .

Quercetin and chlorpromazine were also tested against two PLA2 fractions purified from the plasma of septic_shock patients ; chlorpromazine was then equally potent towards the two PLA2 fractions , whereas quercetin was a potent inhibitor of only one of the two PLA2 fractions (IC50 = 4 microM) .

These data confirm that the observed increase in serum PLA2 activity in septic_shock is due to intravascular release of group II nonpancreatic PLA2 .

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In a prospective study of patients with septic_shock , we have determined the relationship of PLA2 enzyme activity to PLA2 immunoreactivity using radiolabelled E. _ coli phospholipid substrate and an ELISA specific for group II human nonpancreatic PLA2 .

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It was observed that plasminogen bound to hsps 27 , 60 , and 70 .

[Use of ACTH in the treatment of obstetric_and_surgical_shock] .

Despite the success of the anti-coagulant protease protein_C (PC) in treating septic_shock in humans , the signaling pathways used are still unclear .

An early increase in endothelial_protein_C_receptor is associated with excess mortality in pneumococcal_pneumonia with septic_shock in the ICU .

Parathyroid_hormone levels significantly rise in septic_shock .

In conclusion , PTX3 was an early indicator of shock in patients with severe meningococcal_disease that followed a pattern of induction distinct from CRP .

To address this point , PTX3 was measured in plasma from septic_shock patients at day 3 after ICU admission as well as in healthy volunteers (HV) , and the capacity of whole blood cells to secrete PTX3 after inflammatory stimulation was evaluated ex vivo .

We found that a high baseline PTX3 level above the median was significantly associated with the presence of septic_shock , amputation , and 180-day mortality , albeit PTX3 was not an independent predictor of mortality .

CONCLUSIONS : The baseline PTX3 level was an independent predictor for 28-day mortality in patients with septic_shock .

Albumin supplementation was associated with lower levels of PTX3 in patients with septic_shock (P = 0 005) but not in those without shock .
Patients with septic_shock show lower levels of PTX3 when assigned to albumin than to crystalloids .
Baseline plasma PTX3 level was significantly higher in patients with septic_shock (67.3 versus 24.6 ng/mL , p < 0.0001) and in patients who underwent amputation (118.6 versus 43.6 ng/mL , p = 0.019) .
We compared PTX3 , procalcitonin and C-reactive_protein in septic_shock versus nonshock patients and in amputated versus nonamputated patients using the Mann-Whitney U test .
CONCLUSIONS : High PTX3 level is associated with septic_shock , amputation and risk of death in patients with NSTI , but it is not an independent predictor of 180-day mortality in this patient group .
On day 1 , PTX3 levels were higher in septic_shock than in severely septic patients (p = 0.029) .
Pentraxin-3 (PTX3) , an acute-phase protein that belongs to the family of the PTXs , is found elevated in septic_shock and increased in patients with acute_myocardial_infarction .
BACKGROUND : The prognostic value of the acute phase protein Pentraxin_3 (PTX-3) is not well evaluated in patients with septic_shock , which reveal an unacceptably high short - and long-term mortality .
Resistin and NGAL were associated with septic_shock but had limited predictive utility for mortality .
Even after 4 days of renal replacement therapy , plasma from patients with septic_shock plus acute_kidney_injury still showed elevated resistin levels and inhibited neutrophilic-differentiated NB4 cell migration .
Plasma resistin was significantly higher in patients with septic_shock with acute_kidney_injury compared with patients with septic_shock alone .
Here , we studied resistin up to 2 wks after admission in patients with septic_shock and/or severe sepsis .
Raftlin levels were higher in patients with septic_shock , 891.6 (789.7-1,087.8 , n = 30) than in patients with severe sepsis , 681.6 (480.1-819.6 , n = 22) or sepsis , 496.1 (418.1-738.9 , n = 30) , compared with healthy volunteers 364.9 (312.1-392.4 , n = 21) .
Alarmins S100A8 and S100A9 have been shown to be increased after septic_shock .
We studied S-100B in a porcine model of endotoxemic_shock that resembles human Gram-negative septic_shock .
We studied S-100B in a porcine model of endotoxemic_shock that resembles human Gram-negative septic_shock .
We studied the effect of DrotAA on S100B levels in patients with acute septic_shock who presented with increased baseline values of this biomarker .
Adjusting for established risk factors and cardiovascular biomarkers , secretoneurin levels on day 1 were associated with ICU (odds ratio , 2.27 [95 % CI , 1.05-4.93] ; p = 0.04) and 90-day mortality (2.04 [1.02-4.10] ; p = 0.04) in patients with septic_shock , but not severe sepsis without shock .
To test the hypothesis , we measured SDC-1 and VAP-1 levels in 20 patients with septic_shock .
In the septic_shock group SDC-1 correlated on day 1 to SOFA score .
We hypothesize that , in septic_shock , and similar syndromes such as systemic_inflammatory_response_syndrome (SIRS) , Sel-P binds massively to endothelium , causing a drop in Sel-P plasma concentration .
CONCLUSIONS : Lower serum kallistatin level at 24 h was independently associated with 28-day mortality in patients with septic_shock .
Lower serum kallistatin level is associated with 28-day mortality in patients with septic_shock .
High kallistatin levels were also independently associated with a decreased risk of septic_shock , the development of acute_respiratory_distress_syndrome , and positive blood cultures .

Plasma kallistatin levels on day 1 of ICU admission were lower in patients with septic_shock compared with patients with severe sepsis (p = 0.004) .
Plasma corticosteroid-binding_globulin (CBG) concentrations decrease dramatically in patients with septic_shock or burn_injury .
Modeling studies emphasize the significant contribution of albumin deficiency and albumin-bound cortisol under conditions of CBG-deficiency , and identify a synergistic effect by which combined CBG and albumin deficiency contribute to elevation of free cortisol in septic_shock .
CONCLUSIONS : We found extremely low CBG levels in early stage septic_shock and multitrauma .
Antithrombin_III and plasma substitution in septic_shock .
The use of antithrombin_III (ATIII) for disseminated intravascular_coagulation (DIC) during septic_shock .
Numerous uncontrolled clinical studies have reported that antithrombin_III (ATIII) substitution might prevent DIC and death in septic_shock .
Double-blind , placebo-controlled trial of antithrombin_III concentrates in septic_shock with disseminated_intravascular_coagulation .
Since antithrombin_III supplementation has been shown to be beneficial in animal models of septic_shock with disseminated intravascular_coagulation , a controlled study was performed to investigate the effect of antithrombin_III supplementation in fulminant_hepatic_failure .
Since antithrombin_III supplementation has been shown to be beneficial in animal models of septic_shock with disseminated intravascular_coagulation , a controlled study was performed to investigate the effect of antithrombin_III supplementation in fulminant_hepatic_failure .
In 42 patients with septic_shock , 29 of whom underwent substitution with antithrombin_III concentrate and fresh frozen plasma for coagulation_disorders , the proteinase-inhibitor complexes thrombin-antithrombin_III and neutrophil_elastase-alpha 1 proteinase inhibitor , were elevated on admission .
The disturbance of hemostasis in septic_shock : role of neutrophil_elastase and thrombin , effects of antithrombin_III and plasma substitution .
CONCLUSION : Treatment of septic_shock with high-doses of antithrombin_III was effective and safe in patients with an agranulocytosis .
[Therapy with high-doses of antithrombin_III in neutropenic patients with septic_shock] .
Terminal stages of septic_shock are characterized by profound hypocoagulation without potential hypercoagulation , predominance of antithrombin and antiaggregant blood activity with persistent depletion of antithrombin_III and plasminogen .
The study included 42 patients with sepsis , 75 patients with severe sepsis , and 65 patients with septic_shock , who were administered antithrombin_III .
Antithrombin supplementation for anticoagulation during continuous hemofiltration in critically ill patients with septic_shock : a case-control study .
PAI-1 levels greater than 360 micrograms/L on admission predicted the development of a severe septic_shock combined with renal_impairment correctly in 12 of 13 patients (92 %) .
PAI-1 levels were significantly (705.5 ng/ml [131-5788]) higher in septic_shock as in severe sepsis patients (316.5 ng/ml [53-1311] , p = 0.016) and were equal in survivors and non-survivors (342 ng/ml [53-1311] vs. 413 ng/ml [55-5788] , p = 0.231) .
The increase in PAI activity and antigen in septic_shock was accompanied by an increase in tissue-type plasminogen activator antigen and total fibrin (ogen) degradation products and a decrease in alpha (2) - antiplasmin activity (p < 0.006) .
The first results of therapeutic intervention with C1-INH concentrate in septic_shock are promising .
Further evaluation of the serial change of C1INH and the validity of C1INH replacement therapy in patients with septic_shock may lead to a new strategy for sepsis management .

SIRT1 promotes the DNA-bound state of HSF1 through deacetylation of the DNA-binding domain of HSF1 , thereby enhancing the HSR .
BACKGROUND : Oxidative stress is a key feature of sepsis and could be a common pathophysiological pathway between septic_shock and acute_kidney_injury (AKI) Our objective was to evaluate the erythrocyte superoxide dismutase (SOD1) activity as predictor of AKI in patients with septic_shock .
METHODS : We measured the levels of OPN and suPAR for 15 days in forty-three patients , defined a priory as at risk to develop septic_shock .
These results strongly suggest that hyperosmotic_shock activates STAT1 and SHP2 via p38 and its upstream activator MKK6 .
p-KDM3A directly interacts with and is recruited by the transcription factor Stat1 to activate p-KDM3A target genes under heat_shock conditions .
This is a novel example of how multiple activation steps occur under heat_shock , first on the kinases and then the Stat1 and the SWI/SNF chromatin remodeling complex that follows to conduct an auto-regulation based fully activation of the gene .
We show that Jak2 , a Janus kinase specifically associated with the beta subunit of IFNgamma receptor , and PKCepsilon an isoform of the atypical PKC family , are the two dominant kinases responsible for the heat_shock induced phosphorylation on Y701 and S727 of Stat1 .
Stat1 mediates an auto-regulation of hsp90beta gene in heat_shock response .
The results provided the first evidence on the tumor suppressor Stat1 that it could play diverse roles on its target genes under heat_shock that also shed lights on patients with fever or under thermotherapy .
In conclusion , the status and efficacy of Stat1 bound at the GAS of its target gene are pivotal in determining the impact of Stat1 under heat_shock .
In conclusion , the status and efficacy of Stat1 bound at the GAS of its target gene are pivotal in determining the impact of Stat1 under heat_shock .
The result of Stat1 in complex with Stat3 and HSF1 that bound at the GAS to lead a moderate heat_shock induction was designated as an `` intrinsic '' induction of the hsp90alpha gene .
We found that Stat1 regulates the heat_shock induction of its target genes , the hsp90alpha gene in a heat_shock response while the constitutive activity of the gene remains unaffected .
We found that Stat1 regulates the heat_shock induction of its target genes , the hsp90alpha gene in a heat_shock response while the constitutive activity of the gene remains unaffected .
We used RNAi knockdown , point mutations , ChIP and promoter activity assays to study the effect of Stat1 on the heat-shock induction of the hsp90alpha gene under heat_shock conditions .
Stat1 has been known as a regulator of gene expression and a mediator of IFNgamma signaling in mammalian cells , while its effect in a heat_shock response remains unclear .
Diverse effects of Stat1 on the regulation of hsp90alpha gene under heat_shock .
STAT3 protects cardiomyocytes during endotoxic_shock and ischemia and prolongs survival of these cells by activation of antiapoptotic genes like Bcl-2 and c-Fos .
Induction of heat_shock protein 47 synthesis by TGF-beta and IL-1_beta via enhancement of the heat_shock element binding activity of heat_shock transcription factor 1 .
Induction of heat_shock protein 47 synthesis by TGF-beta and IL-1_beta via enhancement of the heat_shock element binding activity of heat_shock transcription factor 1 .
Induction of heat_shock protein 47 synthesis by TGF-beta and IL-1_beta via enhancement of the heat_shock element binding activity of heat_shock transcription factor 1 .
TGF-beta1 levels were decreased in patients with septic_shock (25 + / - 6 pg/ml) as compared with those in normal subjects (37 + / - 2 pg/ml) ($p < 0.05$) .

Furthermore , in septic_shock associated ARDS patients , TGF-b1 levels were significantly higher in nonsurvivors than in survivors [85.23 (78.19-96.30 pg/ml) versus 36.41 (30.21-55.47 pg/ml) , respectively] (p = 0.006) on day 7 of ICU follow-up .
At ICU admission , there were not statistical differences in TGF-b1 concentrations between septic_shock patients with or without ARDS .
The serum thrombomodulin levels at 4 and 8 hours after surgery were higher in the non-cardiogenic_shock group than cardiogenic_shock group (all P < 0.05) .
Septic_shock nonsurvivors had significantly greater mean thrombomodulin concentrations (10.6 + / - 2.2 ng/mL) than septic_shock survivors (5.5 + / - 0.6 ng/mL) (p < .05) and healthy control patients (3.4 + / - 0.2 ng/mL) (p < .01) .
[Septic_shock following flexible transurethral lithotripsy showing favourable response after multidisciplinary treatment mainly composed of recombinant thrombomodulin : a case report] .
The patients with septic_shock demonstrated significantly increased plasma levels of thrombomodulin (p < 0.001) and vWF (p < 0.001) compared with those in healthy controls .
These increased levels of plasma thrombomodulin and vWF in patients with septic_shock decreased significantly after treatment with PMX-F (p < 0.01) .
INTERVENTIONS : Blood samples were obtained for plasma thrombomodulin determinations every 6 hrs for 72 hrs in septic_shock patients and once in healthy control patients .
OBJECTIVES : To test the hypothesis that children diagnosed with septic_shock have increased plasma thrombomodulin values as a manifestation of microcirculatory_dysfunction and endothelial_injury ; to determine whether plasma thrombomodulin concentrations are associated with the extent of multiple_organ_system_failure and mortality .
OBJECTIVES : To test the hypothesis that children diagnosed with septic_shock have increased plasma thrombomodulin values as a manifestation of microcirculatory_dysfunction and endothelial_injury ; to determine whether plasma thrombomodulin concentrations are associated with the extent of multiple_organ_system_failure and mortality .
We speculate that measurement of plasma thrombomodulin concentrations in septic_shock may be a useful indicator of the severity of endothelial damage and the development of multiple_organ_system_failure .
CONCLUSIONS : Pediatric survivors and nonsurvivors of septic_shock have circulating thrombomodulin concentrations 1.5 and 3 times greater than healthy control patients .
Increased circulating thrombomodulin in children with septic_shock .
Successful treatments with polymyxin B hemoperfusion and recombinant human thrombomodulin for fulminant Clostridium_difficile-associated colitis with septic_shock and disseminated_intravascular_coagulation : a case report .
We herein present a case of a 65-year-old woman who developed severe septic_shock following flexible transurethral lithotripsy (f-TUL) showing favourable response after treatment with doripenem and recombinant thrombomodulin (rTM) .
In the present study , transcriptional control was not involved in the induction of TSP1 by heat_shock .
When analyzed for AKI etiology , highest [TIMP-2] [IGFBP7] values were found in patients with septic_shock (P < 0.001 vs. non-AKI I+II) .
From the PPI network , the top 10 hub genes , which are all upregulated DEGs in the septic_shock children , were identified as GAPDH , TNF , EGF , MAPK3 , IL-10 , TLR4 , MAPK14 , IL-1b , PIK3CB , and TLR2 .
Heat_shock up-regulates TLR9 expression in human B cells through activation of ERK and NF-kappaB signal pathways .
In this study , we investigated regulation of TLR9 expression and function in human B cell line RPMI8226 by heat_shock .

The consequences of uncontrolled TLR9 activation can be detrimental for the host , contributing to the pathogenesis of bacterial septic_shock or autoimmune_diseases , such as systemic_lupus_erythematosus .
Plasma levels of tumor_necrosis_factor-alpha (TNF-alpha) , interleukin_1_beta (IL-1_beta) IL-2 , and IL-6 were significantly higher in septic_shock than in sepsis without shock .
Polymorphisms in the tumor_necrosis_factor-alpha gene at position -308 and the inducible 70 kd heat_shock protein gene at position +1267 in multifetal pregnancies and preterm premature rupture_of_fetal_membranes .
Tumor_necrosis_factor is a cytokine made by macrophages , monocytes and T cells that has been formed to play an important role in shock , cachexia and inflammation .
Tumor_necrosis_factor as a mediator of shock , cachexia and inflammation .
Several lines of evidence implicate tumor_necrosis_factor (TNF) , a cytokine produced by monocytes-macrophages , in the systemic manifestations of shock induced by Gram-negative bacteria .
Plasma levels of tumor_necrosis_factor-alpha (TNF-alpha) , interleukin_1_beta (IL-1_beta) IL-2 , and IL-6 were significantly higher in septic_shock than in sepsis without shock .
Indeed , TNF-alpha mRNA expression decreases in cortex , pituitary and liver immediately after IS .
Therefore , we investigated the correlation between the dynamic change of HSP expression and the levels of apoptosis induced by TNF-alpha after HS .
TNF-alpha , IL-1_alpha , IL-6 and ICAM-1 expression in human keratinocytes stimulated in vitro with Escherichia_coli heat-shock_proteins .
Transient overexpression of HSP72 was achieved using an adenoviral vector (Advhsp72) and apoptosis was induced by heat_shock , tumour necrosis factor (TNF) - alpha with cycloheximide (CHX) , lipopolysaccharide (LPS) with TNF-alpha and verocytotoxin (VT) .
Moreover , temperature at 40 degrees C is sufficient to induce heat_shock and attenuate both TNFalpha and IL-1 expression .
These data suggest that lipoic_acid does not have a proinflammatory role and that heat_shock_acts as an anti-inflammatory agent by downregulating TNF-a expression in C2C12 myotubes .
Using a radioimmunoassay , we measured the TNF concentrations in the sera of 7 patients with severe infections without shock , 16 patients with septic_shock and 8 patients with non-septic_shock .
Whether the increase of circulating TNF levels is specific to septic_shock as compared to sepsis without shock or to non-septic_shock is still unclear .
Several lines of evidence implicate tumor_necrosis_factor (TNF) , a cytokine produced by monocytes-macrophages , in the systemic manifestations of shock induced by Gram-negative bacteria .
The value of 250 pg/ml seems to be critical : no patient without shock had TNF levels above 250 and all the patients who died early during the first 24 h) had TNF levels above 250 .
During septic_shock (n = 7) , TNF levels were significantly higher (m = 354 + / - 131 pg/ml) than during sepsis without shock (n = 8 ; m = 145 + / - 35 pg/ml) (p less than 0.0005) .
To confirm the involvement of TNF in human septic_shock , serum TNF levels were measured in 10 adult patients admitted to the intensive care unit for sepsis with or without shock .
To confirm the involvement of TNF in human septic_shock , serum TNF levels were measured in 10 adult patients admitted to the intensive care unit for sepsis with or without shock .
Tumour_necrosis_factor (TNF) is a probable mediator of endotoxic_shock and infusion of this monokine into animals causes multi-organ_failure that shares features with FHF .
Treatment of septic_shock with antibodies to tumour_necrosis_factor .
Overproduction of tumour_necrosis_factor (endotoxin , lipopolysaccharide) leads to septic_shock .
BACKGROUND : Tumor_necrosis_factor-alpha (TNF-alpha) , a proinflammatory cytokine with potent negative inotropic properties , is elaborated in septic_shock , acute myocarditis , reperfusion_injury , and congestive_heart_failure .

Tumor_necrosis_factor-alpha (TNFalpha) is a potent pro-inflammatory cytokine that plays a major role in the pathogenesis of acute and chronic inflammatory_disorders such as septic_shock and arthritis , respectively .
Tumor_necrosis_factor_alpha (TNF_alpha) has been implicated as one of the major mediators of the gram-negative septic_shock_syndrome .
Tumor_necrosis_factor_alpha (TNF_alpha) a pro-inflammatory cytokine is an endogenous mediator of septic_shock , inflammation , anti-viral responses and apoptotic cell death .
Tumor_necrosis_factor_alpha and interleukin_1beta are responsible for in vitro myocardial_cell_depression induced by human septic_shock serum .
Tumor_necrosis_factor_alpha (TNF_alpha) and interleukins (IL) are the principal cytokines involved in the clinical and biological manifestations of septic_shock .
Tumor_necrosis_factor_alpha is a mediator of septic_shock and death , and it exerts its biologic effects by interacting with 2 receptors , TNF-R1 and TNF-R2 .
CONTEXT : Tumor_necrosis_factor_alpha (TNF-alpha) is believed to be a cytokine central to pathogenesis of septic_shock .
BACKGROUND : Tumor_necrosis_factor_alpha (TNF-alpha) is an important mediator of septic_shock .
Plasma levels of tumor_necrosis_factor-alpha (TNF-alpha) , interleukin_1_beta (IL-1_beta) IL-2 , and IL-6 were significantly higher in septic_shock than in sepsis without shock .
Elevated serum levels of the type I and type II receptors for tumor_necrosis_factor-alpha as predictive factors for ARF in patients with septic_shock .
We conducted a prospective study with controls in the National Taiwan University Hospital intensive care unit to compare plasma levels of tumor_necrosis_factor_alpha (TNF-alpha) , interleukin_6 (IL-6) , and circulating intercellular adhesion molecule 1 (cICAM-1) with clinical physiologic parameters in the outcome of patients with septic_shock .
OBJECTIVES : In septic_shock , the principal source of increased plasma concentrations of tumor_necrosis_factor_alpha (TNF) is considered to be the macrophage .
The major cytokines involved in septic_shock are tumor_necrosis_factor_alpha (TNF) and interleukin-1 _ (IL-1) .
The level of tumor_necrosis_factor_alpha (TNF_alpha) , a monokine implicated in mediating septic_shock , is elevated in the blood of some patients with sepsis .
Endotoxin (bacterial lipopolysaccharide [LPS]) causes fatal septic_shock via the Toll-like_receptor_4 (TLR-4) protein present on innate immunity effector cells , which activates nuclear_factor_kappa_B (NF-kappaB) , inducing proinflammatory cytokines , including tumor_necrosis_factor_alpha (TNF-alpha) .
Genotyping of tumor_necrosis_factor_alpha (TNF-alpha) has become an important procedure in the selection of high-risk population of septic_shock and prevention from death due to septic_shock .
Genotyping of tumor_necrosis_factor_alpha (TNF-alpha) has become an important procedure in the selection of high-risk population of septic_shock and prevention from death due to septic_shock .
PIC pretreatment before septic_shock resulted in augmented tumor_necrosis_factor_alpha and interleukins 6 and 10 and heightened lethality compared with septic_shock alone .
Human tumor_necrosis_factor_alpha (hTNFalpha) , a pleiotropic cytokine with activities ranging from host defense mechanisms in infection_and_injury to severe toxicity in septic_shock or other related diseases , is a promising target for drug screening .
[Effect of hydrocortisone_sodium_succinate on serum tumor_necrosis_factor-a and interleukin-10 in septic_shock] .
Tumor_necrosis_factor is a key mediator of the septic_shock_syndrome , and its secretion by monocytes is induced by endotoxin .

Tumor_necrosis_factor (TNF-alpha) and nitric_oxide (NO) are important vasoactive mediators of septic_shock .
Tumor_necrosis_factor in septic_shock and multiple system trauma .
Tumor_necrosis_factor (TNF) mediates a wide variety of disease states including septic_shock , acute_and_chronic_inflammation , and cachexia .
BACKGROUND : Tumor_necrosis_factor (TNF) is a cytokine implicated in many disease states , including septic_shock and transplant rejection .
Tumor_necrosis_factor (TNF-alpha) has been implicated as a principal mediator in the pathogenesis of septic_shock .
Tumor_necrosis_factor (TNF) , a peptide produced by macrophages in response to endotoxin , has been implicated as a mediator of septic_shock .
Tumor_necrosis_factor , a mononuclear phagocyte-derived peptide produced in response to lipopolysaccharide , has been shown to mediate certain aspects of septic_shock and multiple_organ_failure resulting from gram-negative septicemia .
Tumor_necrosis_factor (TNF) and interleukin-I (IL-I) were found to be responsible for most of the symptoms of infectious_diseases , from fever to septic_shock .
Tumor_necrosis_factor (TNF) is a cytokine produced by macrophages which mediates septic_shock .
Tumor_necrosis_factor (TNF) induced by bacterial lipopolysaccharide (LPS) was shown to have an important role in precipitation of septic_shock and disseminated_intravascular_clotting (DIC) .
Tumor_necrosis_factor (TNF) is an endogenously produced cytokine that plays a critical role in mediating septic_shock and multi-organ_failure , but previous studies of the role TNF in disease have not examined its role in mucosal_disease processes .
Tumor_necrosis_factor (TNF) has been implicated in the development and pathogenicity of infectious_diseases and autoimmune_disorders , such as septic_shock and arthritis .
OBJECTIVE : Tumor_necrosis_factor (TNF) - alpha administration in large amounts can induce a state of shock similar to that observed in patients suffering from septic_shock .
OBJECTIVE : To review the animal and human data defining the role of tumor_necrosis_factor (TNF) in the pathogenesis of the septic_shock_syndrome , the systemic_inflammatory_response_syndrome , and related pathologic states .
We addressed the direct and indirect effects of tumor_necrosis_factor (TNF) on endothelial cells that can be relevant for the pathogenesis of septic_shock , with particular attention to the acute_respiratory_distress_syndrome (ARDS) and to cerebral_malaria (CM) .
The present study utilized an in vitro myocardial cell assay to examine the role of various human recombinant cytokines , including tumor_necrosis_factor (TNF) alpha and interleukin (IL) 1beta , in depression of cardiac myocyte contractile function induced by serum from humans with septic_shock .
OBJECTIVE : To review the preclinical evidence for the role of tumor_necrosis_factor (TNF) in the pathogenesis of septic_shock and to assess the preclinical efficacy of anti-TNF therapies for this clinical problem .
Pentoxifylline inhibits tumor_necrosis_factor production in septic_shock .
In a study of serum levels of tumor_necrosis_factor (TNF_alpha) and interleukin-1_beta (IL-1_beta) in patients developing sepsis in the ICU , high TNF_alpha levels were found in patients with septic_shock .
The role of tumor_necrosis_factor / cachectin in septic_shock .
Orchestration of septic_shock by cytokines : the role of cachectin (tumor_necrosis_factor) .

The role of tumor_necrosis_factor (TNF-alpha) in the pathogenesis of septic_shock has been assessed by daily measurements of serum TNF-alpha levels in 60 patients admitted to the medical intensive care unit .
We assayed serial plasma samples from 86 patients , who were enrolled in a prospective randomized trial of the effects of methylprednisolone (MPSS) in septic_shock , for the presence of cytokine tumor_necrosis_factor (TNF) using an enzyme-linked immunosorbent assay .
[Specificity and serum concentrations of tumor_necrosis_factor in septic_shock] .
While the production of tumor_necrosis_factor (TNF) and interleukin-6 (IL-6) in septic_shock and other inflammatory states is well established , the role of interleukin-8 (IL-8) , a recently described neutrophil chemoattractant and activator , has yet to be fully elucidated .
The objective of the present observational study was to establish the time-dependent relation between plasma cytokines interleukin _ (IL) -10 , transforming_growth_factor _ (TGF) - beta1 , tumor_necrosis_factor (TNF) - alpha and monocyte HLA-DR expression in 38 adult patients with septic_shock .
METHODS : Patients with septic_shock who were enrolled into the placebo limb of the North American study of the safety and efficacy of murine monoclonal antibody to tumor_necrosis_factor for the treatment of septic_shock (NORASEPT II) were analyzed .
METHODS : Patients with septic_shock who were enrolled into the placebo limb of the North American study of the safety and efficacy of murine monoclonal antibody to tumor_necrosis_factor for the treatment of septic_shock (NORASEPT II) were analyzed .
North American study of the safety and efficacy of murine monoclonal antibody to tumor_necrosis_factor for the treatment of septic_shock .
Although tumor_necrosis_factor (TNF) is a major mediator of endotoxic_shock , the normal function of TNF that has preserved this protein throughout mammalian evolution remains unknown .
In particular , T-cell-derived TNF-alpha plays a critical role in autoimmune_inflammation and superantigen-induced septic_shock .
In this study , we present evidence indicating that pretreatment of human PMN with a prototype formylated peptide such as fMLP results in the inhibition of TNF-alpha secretion , a key molecule that plays a central role in the pathogenesis of septic_shock .
Elimination of circulating TNF-alpha is pathogenetically perspective in respect to therapy of septic_shock .
Tumor_necrosis_factor (TNF-alpha) and nitric_oxide (NO) are important vasoactive mediators of septic_shock .
MEASUREMENTS AND MAIN RESULTS : High concentrations of circulating TNF-alpha and IL-6 were found in patients with septic_shock .
CONCLUSIONS : In septic_shock patients , high amounts of circulating TNF-alpha and IL-6 are found and then correlate with fatal outcome .
When septic_shock patients were compared with traumatized patients resuscitated from hemorrhagic_shock , the former had much higher concentrations of both TNF-alpha and IL-6 throughout the study period ($p < .01$ to $p < .00001$) .
In septic_shock patients , changes in both TNF-alpha and IL-6 were correlated with outcome , higher values being found in patients likely to die .
During the whole study period , nonsurvivor septic_shock patients maintained higher TNF-alpha concentrations than nonsurvivor trauma patients ($p < .001$) .
At study entry , TNF-alpha concentrations were higher in nonsurvivor septic_shock than in nonsurvivor trauma patients (42 + / - 7 vs 13 + / - 2 pg/mL ; $p < .001$) .
We conducted a prospective study with controls in the National Taiwan University Hospital intensive care unit to compare plasma levels of tumor_necrosis_factor_alpha (TNF-alpha) , interleukin_6 (IL-6) ,

and circulating intercellular adhesion molecule 1 (cICAM-1) with clinical physiologic parameters in the outcome of patients with septic_shock .
The median level of TNF-alpha was found to be significantly higher in infants suffering from sepsis (154 pg/mL) particularly in those with septic_shock (242.5 pg/mL) as compared to healthy controls (61.5 pg/mL) ($p < 0.001$) .
The most relevant clinical results are the therapeutic benefits of PTX in attenuating the effects of tumor_necrosis_factor-alpha (TNF-alpha) in conditions such as septic_shock .
MEASUREMENTS AND MAIN RESULTS : TNF-alpha values were highest in the acute phase of septic_shock (53 to 131 pg/mL during septic_shock) , while patients with bacterial_pneumonia had intermediate concentrations (32 pg/mL) .
In addition to IL-6 , TNF-alpha was proved to be the mediator that orchestrates the hemodynamic and tissue_injury in septic_shock .
Animal studies have shown that the septic_shock to endotoxin challenge can be prevented by pretreatment with monoclonal antibody against TNF-alpha .
The capacity of GH to inhibit LPS-induced TNF-alpha production by monocytes without altering other pathways leading to TNF-alpha production may be of potential relevance in septic_shock , since GH is available for clinical use .
Patients with septic_shock demonstrated significantly decreased TNF-alpha and IL-1beta release as compared with normal subjects in response to LPS .
Significant decreases in TNF-alpha release were found in the patients with septic_shock after PMA stimulation .
In response to SEB , patients with sepsis and patient with septic_shock demonstrated significantly decreased release of TNF-alpha and IL-1beta .
As TNF-alpha is considered a proximal mediator in the cascade leading to septic_shock , we evaluated the ability of PTXF to attenuate the cardiovascular manifestations of sepsis secondary to an infusion of group_B_beta-hemolytic_streptococci (GBS) .
CONCLUSIONS : INTERSEPT provides additional clinical data implicating TNF-alpha as an integral mediator of septic_shock .
These results suggest that TNF-alpha alone is not sufficient to induced noticeable MNC procoagulant activity , at least , in the early stage of this septic_shock model .
Although the method is far too slow for any clinical routine work , our results suggest that the presence of elevated serum TNF-alpha levels could be considered a sensitive and specific test for predicting septic_shock and its clinical outcome .
Tumor_necrosis_factor (TNF-alpha) has been implicated as a principal mediator in the pathogenesis of septic_shock .
Cortisol , tumor_necrosis_factor-alpha (TNF-alpha) , and interleukin-6 (IL-6) levels were measured before and after infusion of low (1 microgram) and standard doses (250 micrograms) of adrenocorticotropic_hormone (ACTH) within 24 hours of the diagnosis of septic_shock .
Interleukins (IL) -1 beta , -6 , -8 , and tumour necrosis factor alpha (TNF-alpha) have been implicated as mediators of septic_shock , with circulating leucocytes being considered a major source for their release .
OBJECTIVES : To determine the safety of a `` humanized '' antibody to human anti-tumor_necrosis_factor-alpha (TNF-alpha) in patients with septic_shock , and to examine the pharmacokinetics , immune response , and influence of the antibody on cytokine concentrations in this patient group .
CONCLUSIONS : The humanized anti-TNF-alpha antibody , CDP571 , is well tolerated and able to cause a dose-dependent reduction in circulating TNF-alpha concentrations in patients with septic_shock .

BACKGROUND : Tumor_necrosis_factor-alpha (TNF-alpha) , a proinflammatory cytokine with potent negative inotropic properties , is elaborated in septic_shock , acute myocarditis , reperfusion_injury , and congestive_heart_failure .
Higher level of serum TNF-alpha was significantly associated with the occurrence of septic_shock , but not of fatal outcome .
CONCLUSIONS : Higher levels of serum TNF-alpha are associated with the occurrence of septic_shock .
Thus , SMC both respond to both TNF_and_lymphotoxin and can produce TNF-alpha , a cytokine with numerous effects on vascular cells of potential significance in the pathophysiology of septic_shock and other inflammatory conditions .
The role of tumor_necrosis_factor (TNF-alpha) in the pathogenesis of septic_shock has been assessed by daily measurements of serum TNF-alpha levels in 60 patients admitted to the medical intensive care unit .
The role of tumor_necrosis_factor (TNF-alpha) in the pathogenesis of septic_shock has been assessed by daily measurements of serum TNF-alpha levels in 60 patients admitted to the medical intensive care unit .
Endotoxin (bacterial lipopolysaccharide [LPS]) causes fatal septic_shock via the Toll-like_receptor_4 (TLR-4) protein present on innate immunity effector cells , which activates nuclear_factor_kappa_B (NF-kappaB) , inducing proinflammatory cytokines , including tumor_necrosis_factor_alpha (TNF-alpha) .
In septic_shock , endogenous catecholamines induce beta2-AR downregulation , leading to an increased TNF-alpha release .
Genotyping of tumor_necrosis_factor_alpha (TNF-alpha) has become an important procedure in the selection of high-risk population of septic_shock and prevention from death due to septic_shock .
Genotyping of tumor_necrosis_factor_alpha (TNF-alpha) has become an important procedure in the selection of high-risk population of septic_shock and prevention from death due to septic_shock .
Data for batch adsorption for TNF-alpha was used to estimate the minimum amount of silica required to treat septic_shock .
Prostaglandin_E2 inhibits tumor_necrosis_factor-alpha RNA through PKA type I.
Tumor_necrosis_factor-alpha (TNF-alpha) is a cytokine that may contribute to the pathogenesis of septic_shock , rheumatoid_arthritis , cancer , and diabetes .
Using an in vitro assay , this study sought to determine the effect of TGF-beta1 on myocyte depression induced by TNF-alpha , IL-1beta , and serum with known depressant activity from patients with septic_shock .
Similarly , depressant effects caused by synergistic concentrations of TNF-alpha and IL-1beta and serum from all five patients with septic_shock were prevented by co-incubation with TGF-beta1 .
Tumor_necrosis_factor-alpha (TNF-alpha) , a protein released by activated macrophages , is involved in a wide variety of human diseases including septic_shock , cachexia , and chronic_inflammation .
These results confirm the importance of TNF-alpha in the pathogenesis of septic_shock and suggest a clinical potential for TNFR-IgG as a preventive and therapeutic treatment in sepsis .
The TNF-alpha production capacity of whole blood , which was found to be lower in the septic_shock patients than in healthy subjects , was significantly increased after PMX-DHP .
A significantly larger proportion of children with high IL-1Ra : TNF-alpha and IL-1Ra : IL-6 ratios developed severe disease with septic_shock than those with a low ratios (95.2 % vs. 4.8 % ; 76.2 % vs. 23.8 %) .
The TNF-alpha concentration was higher in those with septic_shock than in those without .
Conversely , due to the participation of TNFs in inflammatory responses , antagonists of TNF-alpha are being considered for the treatment of several conditions , especially septic_shock .

These results suggest that endotoxin and extremely high levels of TNF-alpha and IL-2 , or the simultaneous elevation of IL-1_beta and IL-6 , are related to the onset of septic_shock .
We defined two types of septic_shock from these data , i.e. , endotoxin + TNF-alpha + IL-2 shock and IL-beta + IL-6 shock .
Plasma levels of tumor_necrosis_factor-alpha (TNF-alpha) , interleukin_1_beta (IL-1_beta) IL-2 , and IL-6 were significantly higher in septic_shock than in sepsis without shock .
Specific polymorphisms of the TNF-alpha and TNF-beta genes (TNF2 and LTA + 250 , respectively) have been suggested to correlate with higher mortality in septic_shock .
Pneumococcal septic_shock plasmas with significantly higher levels of HSP70 ($P < 0.05$) did not induce TNF-alpha secretion in the monocytes .
Mononuclear phagocytes play a pivotal role in the progression of septic_shock by producing tumor_necrosis_factor-alpha (TNF-alpha) and other inflammatory mediators in response to lipopolysaccharide (LPS) from Gram-negative bacteria .
Plasma level of TNF-alpha or sTNFR was of little value in predicting the occurrence of early septic_shock in ASP .
Accordingly , in septic_shock caused by either GBS or Gram-negative bacteria , complete inhibition of TNF-alpha release may require treatment with drugs or drug combinations capable of inhibiting multiple activation pathways .
In septic_shock , myositis is thought to be mediated by pro-inflammatory cytokines such as tumour necrosis factor-alpha (TNF-alpha) , interleukin-8 (IL-8) and interleukin-6 (IL-6) but this has never previously been studied in MCD .
Moreover , TNF-alpha responses obtained with high-dose lipopolysaccharide were significantly greater in cells from patients who subsequently survived septic_shock ($n = 13$; median value 1392 pg/ml ; range 592-2048 pg/ml) than in cells from non-survivors ($n = 7$; median value 708 pg/ml ; range 520-1344 pg/ml) .
Levels of TNF-alpha , interleukin-1_beta and interleukin-10 were significantly higher ($P < 0.05$) when cell stimulation was delayed for 16 h , indicating a functional down-regulation of cells during septic_shock .
Tumor_necrosis_factor-alpha (TNF-alpha) is one of the major mediators produced in activated macrophages which contribute to the circulatory failure associated with septic_shock .
In the peritonitis group with subsequent septic_shock , TNF-alpha , sTNF-alpha RI + RII IL-1beta , IL-8 , IL-10 , and nitrate were significantly increased before the onset of septic_shock .
In the peritonitis group with subsequent septic_shock , TNF-alpha , sTNF-alpha RI + RII IL-1beta , IL-8 , IL-10 , and nitrate were significantly increased before the onset of septic_shock .
CONCLUSION : Monocytes from patients with septic_shock exhibit persistent IL-10 release at a time when TNF-alpha release is downregulated .
In conclusion , LPS-induced ex vivo TNF-alpha and G-CSF cytokine release by monocytes is regulated differentially in patients with septic_shock .
Ex vivo LPS-inducible release of granulocyte_colony-stimulating_factor (G-CSF) was upregulated and that of TNF-alpha was downregulated in patients with septic_shock , regardless whether they survived or died .
It was reported that TNF-alpha has numerous actions in diseases such as inflammation , autoimmunity , infectious_diseases , septic_shock and many types of cancer [1 , 2] .
MEASUREMENTS AND MAIN RESULTS : Blood sample was obtained 24 hrs after intensive_care_unit (ICU) admission or within 2 hrs after the onset of septic_shock to determine the plasma TNF-alpha level and to analyze the genotype of the biallelic polymorphism of the TNF-alpha .
It has been postulated that production of TNF-alpha is central to the pathogenesis of septic_shock induced by group B Streptococcus (GBS) .

TNF-alpha , implicated in the pathophysiology of septic_shock , is capable of inducing adult_respiratory_distress_syndrome (ARDS) in experimental animals and humans .
The pathogenesis of septic_shock is mainly due to unregulated tumour_necrosis factor-alpha (TNF-alpha) production .
TNF-alpha is involved in virtually all features of septic_shock and multiple_organ_failure .
OBJECTIVES : To determine the predictive value of early determination of tumor_necrosis_factor (TNF) - alpha , TNF-alpha 1 and 2 soluble receptors (sTNFR1 and sTNFR2) and endothelin-1 (ET-1) for mortality in patients with septic_shock .
CONCLUSIONS : Increased levels of TNF-alpha were consistently higher at all time-points in nonsurvivors with septic_shock .
The neonates with septic_shock had five fold increase in TNF-alpha levels (2262 + / - 605.8 pg/ml) as compared to those without shock (738.8 + / - 728.8 pg/ml) .
TNF-alpha and IL-6 concentrations decreased significantly between the first and third days of septic_shock (p = .0001) , whereas IL-1 concentrations remained low .
CONCLUSIONS : The systemic release of TNF-alpha and IL-6 during septic_shock caused by generalized peritonitis was maximal on day 1 and decreased rapidly during the next days .
CONCLUSIONS : HUVECs are capable of producing TNF-alpha after proinflammatory cytokine stimulation and may therefore contribute to the increased amount of TNF-alpha found in pathologic states such as septic_shock .
CONTEXT : Tumor_necrosis_factor_alpha (TNF-alpha) is believed to be a cytokine central to pathogenesis of septic_shock .
Association of TNF2 , a TNF-alpha promoter polymorphism , with septic_shock susceptibility and mortality : a multicenter study .
Although circulating TNF-alpha and IL-1beta are both often elevated in septic_shock , it remains unknown whether TNF-alpha or IL-1beta are the factors induced during sepsis that directly depress human myocardial function , and if so , whether the combination synergistically depresses myocardial function .
Although circulating TNF-alpha and IL-1beta are both often elevated in septic_shock , it remains unknown whether TNF-alpha or IL-1beta are the factors induced during sepsis that directly depress human myocardial function , and if so , whether the combination synergistically depresses myocardial function .
To evaluate the prognostic value in septic_shock , PCT levels were repeatedly determined and compared with tumour_necrosis factor-alpha (TNF-alpha) - and interleukin _ (IL) -6 bioactivity as well as with C-reactive_protein (CRP) serum levels .
BACKGROUND : Tumor_necrosis_factor_alpha (TNF-alpha) is an important mediator of septic_shock .
Western blot analysis of bile or intestinal fluid from patients with septic_shock or systemic_inflammatory_response_syndrome , using antibodies to TNF-alpha , IL-1_alpha and IL-1_beta .
All these results suggest that a possible mechanism for TNF-alpha production in endotoxic_shock is the increase in gene transactivity induced by LPS in monocytes/macrophages , and that p38 MAPK signal pathway participates in the regulation of TNF-alpha gene expression induced by LPS .
We also revealed the marked elevation of plasma CNP concentration in patients with septic_shock , in which TNF_alpha plays a significant part .
Tumor_necrosis_factor_alpha (TNF_alpha) a pro-inflammatory cytokine is an endogenous mediator of septic_shock , inflammation , anti-viral responses and apoptotic cell death .
A review of the interactions of TNF_alpha with macrophages , neutrophils and endothelium underlines the key role of TNF_alpha in 3 important events of septic_shock : neutrophil adherence to

endothelium , capillary_leak_syndrome , and development of disseminated intravascular_coagulopathy .
CONCLUSIONS : TNF_alpha is a major mediator involved in the pathogenesis of septic_shock and its decrease was significantly associated with a favourable outcome .
In a study of serum levels of tumor_necrosis_factor (TNF_alpha) and interleukin-1_beta (IL-1_beta) in patients developing sepsis in the ICU , high TNF_alpha levels were found in patients with septic_shock .
Although IL-1_beta was never found , TNF_alpha was most often observed in the serum at a level under 100 pg/mL except during septic_shock .
Monoclonal antibody to TNFalpha in septic_shock .
In our previous study , it was found that PMMA-CHDF could efficiently remove various pro-inflammatory cytokines such as TNFalpha , IL-6 and IL-8 from the bloodstream , resulting in early recovery from septic_shock .
TNFalpha was the only variable being higher upon advent of septic_shock compared with patients without SIRS and upon presentation of SIRS , sepsis , and severe sepsis (p of comparisons with all subgroups < 0.0001) .
Tumor_necrosis_factor-alpha (TNFalpha) is a potent pro-inflammatory cytokine that plays a major role in the pathogenesis of acute and chronic inflammatory_disorders such as septic_shock and arthritis , respectively .
TNFalpha plays a role in the pathogenesis of septic_shock , inflammatory diseases , autoimmune_diseases , graft rejection reaction , acute , and chronic respiratory_inefficiency among others .
Continuous veno-venous hemofiltration improves hemodynamics in septic_shock with acute_renal_failure without modifying TNFalpha and IL6 plasma concentrations .
In the absence of septic_shock , there was a significant trend to greater T1RF in patients with LTalpha +250 GG (TNFalpha hyposecretor) genotype (p = 0.03) .
INTERPRETATION : In human septic_shock we found that iNOS activity is compartmentalised at the very site of infection and parallels expression of TNFalpha and IL-1beta .
Because TNFalpha plays a central role in various inflammatory diseases such as endotoxic_shock , multiple_sclerosis , cerebral_malaria , and various autoimmune conditions , the down-regulatory effect of VIP/PACAP may have a significant therapeutic potential .
Increased IL-1b , IL-6 , IL-8 , IL-10 , TNF-a and decreased C4 d , C5a and iC3b levels were associated with septic_shock , coma and mortality .
Methods The serum level of tumor_necrosis_factor_a (TNF-a) was examined by ELISA in 5 healthy volunteers and 5 patients with septic_shock .
Furthermore , fever might therefore occur in the absence of a septic_shock response because of the inhibiting effect of PGE2 on TNF-a production .
Serum TNF-a level is elevated in some pathological states such as septic_shock , graft rejection , HIV_infection , neurodegenerative_diseases , rheumatoid_arthritis and cancer .
OBJECTIVE : To study the effects of Ringer 's sodium_pyruvate solution on tumor_necrosis_factor-a (TNF-a) and interleukin-6 (IL-6) upon septic_shock .
Individuals with the TNF-a 308 rs1800629 A allele (adjusted OR , 2.96 ; 95 % CI , 1.30-7.87) or the IL-6 rs1800795 C allele (adjusted OR , 1.87 ; 95 % CI , 1.03-3.61) had a higher prevalence of septic_shock .
CONCLUSION : In a dose-dependent manner , CHR may inhibit increased permeability of vascular endothelial cells induced by septic_shock patient 's serum , its underlying mechanism may be related to inhibition of the effect of TNF-a .
In conclusion , TNF-a and IL-10 are involved in myocardial_dysfunction accompanying septic_shock in children , and TNF-a is associated with mortality .

However , inhibition of TNF-a with a high dose of a TNF-receptor fusion protein in patients with septic_shock worsened patient survival .
Moreover , we showed that TPO negatively modulates myocardial contractility by stimulating its receptor c-Mpl on cardiomyocytes and the subsequent production of NO , and it mediates the cardiodepressant activity exerted in vitro by serum of septic_shock patients by cooperating with TNF-a and IL-1b .
Plasma total nitrite (nitrites and nitrates) , cytokines like tumour_necrosis_factor-a (TNF-a) and plasma lactate were measured to assess inflammatory activity and severity of septic_shock .
OBJECTIVE : We assessed the relationship of the genotype distribution of -308 G _ > A TNF-a polymorphism with regard to the development of sepsis , septic_shock , higher organ dysfunction or mortality in critically_ill patients .
Septic_shock and respiratory_failure in community-acquired_pneumonia have different TNF polymorphism associations .
Our data strengthen the hypothesis that serum values of total TNF determine the extent of hypocholesterolemia during sepsis and septic_shock despite the presence of a high concentration of TNF receptors .
Patients with septic_shock had significantly higher serum TNF and TNF receptor levels compared with healthy controls .
TNF is produced by monocytes/macrophages in response to endotoxin , which may lead to septic_shock .
CONCLUSION : Although IL-10 has an inhibitory effect on the production of cytokines , it is released together with TNF and IL-6 in patients with septic_shock .
PATIENTS AND METHODS : In 11 patients with septic_shock of recent onset , blood was sampled for determinations of TNF , IL-1 , IL-6 , and IL-10 .
This study may provide the basis for a more physiological therapeutic approach to TNF neutralization in septic_shock patients .
We addressed the direct and indirect effects of tumor_necrosis_factor (TNF) on endothelial cells that can be relevant for the pathogenesis of septic_shock , with particular attention to the acute_respiratory_distress_syndrome (ARDS) and to cerebral_malaria (CM) .
NO might , therefore , be a key mediator of haemodynamic_impairment in humans under conditions with known elevations of circulating TNF , such as a septic_shock .
Tumor_necrosis_factor (TNF) mediates a wide variety of disease states including septic_shock , acute_and_chronic_inflammation , and cachexia .
OBJECTIVES : In septic_shock , the principal source of increased plasma concentrations of tumor_necrosis_factor_alpha (TNF) is considered to be the macrophage .
Further studies are needed to determine if pentoxifylline 's ability to lower circulating TNF concentration without altering hemodynamics will improve outcome in septic_shock .
CONCLUSIONS : Pentoxifylline is able to decrease serum TNF but not IL-6 or IL-8 serum concentrations during septic_shock .
BACKGROUND : Tumor_necrosis_factor (TNF) is a cytokine implicated in many disease states , including septic_shock and transplant rejection .
This method may be used to detect early TNF production in disease states such as septic_shock and transplant rejection .
Clearly , further work must be done to resolve the issue of the exact role of TNF in organ injury during septic_shock .
OBJECTIVE : To review the preclinical evidence for the role of tumor_necrosis_factor (TNF) in the pathogenesis of septic_shock and to assess the preclinical efficacy of anti-TNF therapies for this clinical problem .

The major cytokines involved in septic_shock are tumor_necrosis_factor_alpha (TNF) and interleukin-1 _ (IL-1) .
The highest TNF levels were found in those newborns with septic_shock , particularly in those who died .
As TNF and PAF are thought to be involved in the development of septic_shock and adult respiratory_distress_syndrome , we hypothesize that high-dose Ara-C may be associated with cytokine release .
Tumor_necrosis_factor-alpha (TNF) has been implicated in the pathogenesis of a variety of human diseases including septic_shock , cachexia , graft-versus-host_disease and several autoimmune_diseases .
In septic_shock tumor_necrosis_factor (TNF) leads to increased nitric_oxide (NO) production by induction of NO synthase .
Administration of the complex prepared by various lipopolysaccharides produced significantly less quantities of TNF in the septic_shock model .
BACKGROUND : The overproduction of tumor_necrosis_factor-alpha (TNF) plays a key role in virtually every experimental model of septic_shock , which has led to the development of several therapies that target TNF and other cytokines in clinical_sepsis .
BACKGROUND : The overproduction of tumor_necrosis_factor-alpha (TNF) plays a key role in virtually every experimental model of septic_shock , which has led to the development of several therapies that target TNF and other cytokines in clinical_sepsis .
In contrast , septic_shock and cachexia may result from either acute or chronic systemic activation of monocytes , resulting in the widespread release of TNF secretory component into the circulation of the affected individual .
Since similar physiological changes have been reported after endotoxin injection , our data support the suggestion that TNF production is a critical factor in the development of septic_shock .
Tumor_necrosis_factor_alpha (TNF) , a monokine produced by mononuclear cells in response to bacterial endotoxin (LPS) , creates a syndrome similar to septic_shock in animal models .
From the PPI network , the top 10 hub genes , which are all upregulated DEGs in the septic_shock children , were identified as GAPDH , TNF , EGF , MAPK3 , IL-10 , TLR4 , MAPK14 , IL-1b , PIK3CB , and TLR2 .
Tumor_necrosis_factor / cachectin (TNF) is a mediator of the septic_shock state , which can modulate hemostatic properties of the vessel wall .
Tumor_necrosis_factor-alpha (TNF) has received particular attention because of its central role in septic_shock and more recent work has shown its participation in transplant immunology .
Whereas TNF increased chemotaxis at low concentrations in the presence of 1 % ZAS , high concentrations of TNF similar to levels detected in septic_shock caused a decrease in chemotaxis that might contribute to retaining PMN in sites of inflammation .
Whereas TNF increased chemotaxis at low concentrations in the presence of 1 % ZAS , high concentrations of TNF similar to levels detected in septic_shock caused a decrease in chemotaxis that might contribute to retaining PMN in sites of inflammation .
Tumor_necrosis_factor (TNF) , a peptide produced by macrophages in response to endotoxin , has been implicated as a mediator of septic_shock .
The use of specific antibodies to inhibit production of TNF , or other agents to antagonise the toxic effects of TNF may have clinical relevance in counteracting septic_shock and the clinical manifestations of TNF in inflammatory and neoplastic_disease .
We assayed serial plasma samples from 86 patients , who were enrolled in a prospective randomized trial of the effects of methylprednisolone (MPSS) in septic_shock , for the presence of cytokine tumor_necrosis_factor (TNF) using an enzyme-linked immunosorbent assay .

Serum cachectin/tumor _ necrosis_factor (TNF) , a cytokine implicated in the pathogenesis of septic_shock , may appear in the circulation during serious infection , but the frequency of detection of elevated serum levels during protracted critical burn_injury is unknown .
The circulating TNF level , determined upon admission , appears to be neither specific nor predictive of the outcome of septic_shock .
Whether the increase of circulating TNF levels is specific to septic_shock as compared to sepsis without shock or to non-septic_shock is still unclear .
In contrast , persistently high levels of circulating TNF seem to be well correlated with a poor prognosis , since 5 out of 6 patients with elevated TNF values died of septic_shock .
In contrast , persistently high levels of circulating TNF seem to be well correlated with a poor prognosis , since 5 out of 6 patients with elevated TNF values died of septic_shock .
Pretreatment with monoclonal antibodies against TNF prevents the occurrence of septic_shock after endotoxin administration .
Moreover , high concentrations of TNF have been found in patients suffering from septic_shock .
Giving TNF mimicks the clinical and biological patterns of septic_shock .
On the other hand , systemic or local TNF overexpression is typical of such pathological states as rheumatoid_arthritis , psoriasis , Crohn 's _ disease , septic_shock , and multiple_sclerosis .
Recent studies indicate that overproduction of TNF in septicaemia is a critical step in triggering septic_shock and multiple organ damage .
Glucocorticoids almost completely inhibit the synthesis by isolated macrophages of cachectin/tumor _ necrosis_factor (TNF) , a cytokine implicated as a major endogenous mediator of septic_shock .
Thus , in patients with septic_shock due to various gram-negative bacteria , other parameters than the absolute serum concentration of immunoreactive TNF contributed significantly to the prediction of outcome .
Serum concentrations of immunoreactive tumor_necrosis_factor / cachectin (TNF) , interleukin-1_beta (IL-1_beta) , interferon-gamma (IFN_gamma) , and interferon-alpha (IFN_alpha) were prospectively measured in 70 patients with septic_shock to determine their evolution and prognostic values .
Tumor_necrosis_factor (TNF) and interleukin-I (IL-I) were found to be responsible for most of the symptoms of infectious_diseases , from fever to septic_shock .
During septic_shock (n = 7) , TNF levels were significantly higher (m = 354 + / - 131 pg/ml) than during sepsis without shock (n = 8 ; m = 145 + / - 35 pg/ml) (p less than 0.0005) .
To confirm the involvement of TNF in human septic_shock , serum TNF levels were measured in 10 adult patients admitted to the intensive care unit for sepsis with or without shock .
To confirm the involvement of TNF in human septic_shock , serum TNF levels were measured in 10 adult patients admitted to the intensive care unit for sepsis with or without shock .
Recent experiments have demonstrated that TNF plays an important role in the pathogenesis of septic_shock .
Tumor_necrosis_factor (TNF) is a cytokine produced by macrophages which mediates septic_shock .
Tumor_necrosis_factor (TNF) induced by bacterial lipopolysaccharide (LPS) was shown to have an important role in precipitation of septic_shock and disseminated_intravascular_clotting (DIC) .
While the production of tumor_necrosis_factor (TNF) and interleukin-6 (IL-6) in septic_shock and other inflammatory states is well established , the role of interleukin-8 (IL-8) , a recently described neutrophil chemoattractant and activator , has yet to be fully elucidated .
Monoclonal antibody to TNF in severe septic_shock .
TNF , alone or together with endotoxin or IL-1 , is capable of inducing lethal shock and tissue injury resembling that of septic_shock .

The macrophage-derived TNF has been implicated as the most important host mediator in the pathogenesis of septic_shock .
Three important cytokines associated with septic_shock are tumour_necrosis_factor / cachectin (TNF) , interleukin_1 _ (IL-1) and interleukin_6 (IL-6) .
TNF levels greater than 50 pg/ml were noted in six febrile leukemic patients and two bacteria-infected patients (both of whom were complicated by septic_shock) .
On the other hand , TNF can also cause septic_shock , tissue injury and cachexia .
Furthermore , because TNF challenge induced both sustained pulmonary_and_cardiac_injury , TNF may be a common pathway for the multiple organ dysfunction that occurs during septic_shock .
Furthermore , because TNF challenge induced both sustained pulmonary_and_cardiac_injury , TNF may be a common pathway for the multiple organ dysfunction that occurs during septic_shock .
TNF and other cytokines in the metabolism of septic_shock and cachexia .
This resulted in plasma TNF levels considerably greater than those observed in septic_shock .
MEASUREMENTS AND RESULTS : Median IL-1Ra : TNF and IL-1Ra : IL-6 ratios were significantly higher in severe disease with septic_shock than in severe disease without septic_shock and in non severe disease (IL-1Ra : TNF 263 vs. 185 vs. 108 ; IL-1Ra : IL-6 139 vs. 23 vs. 17) .
Tumor_necrosis_factor -- alpha (TNF) , one of the mediators of septic_shock , has a role in the immunopathological complications of several infections .
Antibodies against TNF prevent and reverse these sequelae in animal models of septic_shock , and their use in humans is currently under investigation in clinical trials .
A promising new development in the treatment of this condition is the use of monoclonal antibodies to inactivate two prime mediators that induce the cascade of events that culminate in septic_shock and multiple_organ_failure : bacterial endotoxin and tumor_necrosis_factor (TNF) .
The improvement in cardiac function following anti-TNF antibody administration in patients is in keeping with recent experimental studies indicating the role of TNF in the myocardial_depression characterizing septic_shock .
On the one hand , the cytokine pattern in experimental septic_shock and meningococcal_disease has similarities which include early burst releases of TNF and IL-6 .
In addition to mediating systemic_disease , such as septic_shock , TNF is also produced locally , and can play a critical role in mediating mucosal_disease processes , such as acute_gonococcal_salpingitis .
Tumor_necrosis_factor (TNF) is an endogenously produced cytokine that plays a critical role in mediating septic_shock and multi-organ_failure , but previous studies of the role TNF in disease have not examined its role in mucosal_disease processes .
Because of the implication of TNF in the pathogenesis of septic_shock , we suggest that neutralization or elimination of this cytokine by plasma or blood exchanges could contribute to the treatment of severe forms of this syndrome .
Previous investigations suggest that TNF plays a prominent role in septic_shock and meningococcal_disease_toxicity .
CONCLUSIONS : Results indicate that the TNF gene polymorphisms studied play no part in determination of disease severity or ASP susceptibility ; however , they are both strongly related to the development of septic_shock in ASP .
Tumor_necrosis_factor (TNF) has been implicated in the development and pathogenicity of infectious_diseases and autoimmune_disorders , such as septic_shock and arthritis .
CONCLUSION : Results indicate that TNF gene polymorphisms studied play no part in determination of disease severity or susceptibility to acute_biliary_pancreatitis ; however , TNF2 polymorphism is associated with septic_shock from ASBP .

MATERIAL AND METHODS : The examination of 33 patients with HM and septic_shock included measurement of plasma concentrations of tumor_necrosis factor (TNF) , interleukine-6 (IL-6) , endotoxin , procalcitonin (PCT) 12-24 hours before and each 12 hours after shock ; registration of central hemodynamics parameters , the condition severity by APACHE_II .
Despite the potentially important role that TNF and IL-1beta may play in producing cardiac_dysfunction in human septic_shock , little is known with regard to the basic biochemical mechanism (s) by which bacterial pathogens induce their expression in the heart .
OBJECTIVE : Tumor_necrosis_factor (TNF) - alpha administration in large amounts can induce a state of shock similar to that observed in patients suffering from septic_shock .
In those who developed septic_shock , the TNF2 allele was significantly associated with higher TNF levels .
Although the antitumour activity and mediation of inflammation by TNF could be beneficial to the host , unregulated TNF is now known to be the basis for development of various diseases including septic_shock , the wasting_disease , cachexia , and various inflammatory and/or autoimmune_diseases .
Although the antitumour activity and mediation of inflammation by TNF could be beneficial to the host , unregulated TNF is now known to be the basis for development of various diseases including septic_shock , the wasting_disease , cachexia , and various inflammatory and/or autoimmune_diseases .
Identification of new therapeutic targets for the management of septic_shock remains imperative as all investigational therapies , including anti-tumor_necrosis_factor (TNF) and anti-interleukin (IL) -1 agents , have uniformly failed to lower the mortality of critically ill patients with severe sepsis .
Tumor_necrosis_factor-alpha (TNF) is a pluripotent cytokine that mediates many of the hemodynamic manifestations of endotoxic_shock .
Since heat-stable antigens are present in the circulation of patients with malaria , they may induced the secretion of TNF , a mediator of endotoxic_shock , which could contribute to the pathology of the disease .
Tumour_necrosis_factor (TNF) is a probable mediator of endotoxic_shock and infusion of this monokine into animals causes multi-organ_failure that shares features with FHF .
TNF has been implicated as a mediator of endotoxic_shock , inflammatory_joint_disease , immune_deficiency states , allograft rejection , and in the cachexia associated with malignant_disease and some parasitic_infections .
TNF is now recognized to be the major effector of gram-negative endotoxic_shock .
Although tumor_necrosis_factor (TNF) is a major mediator of endotoxic_shock , the normal function of TNF that has preserved this protein throughout mammalian evolution remains unknown .
Although tumor_necrosis_factor (TNF) is a major mediator of endotoxic_shock , the normal function of TNF that has preserved this protein throughout mammalian evolution remains unknown .
In the paper the possible physiological significance and pathological role of cachectin as a mediator of endotoxic_shock and wasting in chronic_diseases are reviewed .
However , enhanced apoptosis was still observed when TRAIL was added one day after heat_shock .
Nonetheless , when treated with TRAIL for 3 h after release from heat_shock , the human colon_cancer cell line HCT116 is protected from apoptosis whereas the human colon_cancer cell line SW480 is not .
We report that a mild heat_shock , that did not impair cell growth , stimulated TNF-related_apoptosis_inducing_ligand (TRAIL) - mediated apoptosis of leukemic T lymphocytes and promyelocytic cells , but not normal human T lymphocytes .
The TWEAK levels were higher in patients with septic_shock (192.8 230.5 pg/mL) than in controls (84.1 28.7 pg/mL , P = 0.043) .

Enzyme-linked immunoassay was used to measure serum TWEAK levels in 20 patients with septic_shock , all of whom were treated by direct hemoperfusion with a polymyxin B-immobilized fiber column (DHP-PMX) , and in 20 non-septic controls .
Cardiogenic_shock developed in a significantly higher number of patients with elevated serum cTnI levels (33 % vs 5 % , P = .01) .
Receiver-operating characteristics of serum cTnI as a predictor of death in septic_shock were significant .
The VEGF and the soluble Flt-1 levels were more elevated in patients with septic_shock than in controls .
Heat-shock and cadmium_chloride increase the vimentin mRNA and protein levels in U-937 human promonocytic cells .