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Abstract: Sn-doped In2O3 nanowires have been grown on Si via the vapor-liquid-solid mechanism at 800 degrees C and then exposed to H2S between 300 to 600 degrees C. We observe the existence of cubic bixbyite In2O3 and hexagonal SnS2 after processing the Sn:In2O3 nanowires to H2S at 300 degrees C but also cubic bixbyite In2O3, which remains dominant, and the emergence of rhombohedral In2(SO4)3 at 400 degrees C. The resultant nanowires maintain their metallic-like conductivity, and exhibit photoluminescence at 3.4 eV corresponding to band edge emission from In2O3. In contrast, Sn:In2O3 nanowires grown on glass at 500 degrees C can be treated under H2S only below 200 degrees C which is important for the fabrication of Cu2S/Sn:In2O3 core-shell p-n junctions on low-cost transparent substrates such as glass suitable for quantum dot-sensitized solar cells

Abstract: Liver monocytes play a major role in the development of NASH (non-alcoholic steatohepatitis). In inflamed tissues, monocytes can differentiate in both macrophages and dendritic cells. In the present study, we investigated the role of moDCs (monocyte-derived inflammatory dendritic cells) in experimental steatohepatitis induced in C57BL/6 mice by feeding on a MCD (methionine/choline-deficient) diet. The evolution of steatohepatitis was characterized by an increase in hepatic CD45(+)/CD11b(+) myeloid cells displaying the monocyte/macrophage marker F4-80(+). In the early phases (4 weeks of treatment), Ly6C(high)/CD11b(+)/F4-80(+) inflammatory macrophages predominated. However, their frequency did not grow further with the disease progression (8 weeks of treatment), when a 4-fold expansion of CD11b(+)/F4-80(+) cells featuring the fractalkine receptor (CX3CR1) was evident. These CX3CR1(+) cells were also characterized by the combined expression of inflammatory monocyte (Ly6C, CD11b) and dendritic cell (CD11c, MHCII) markers as well as by a sustained TNFalpha (tumour necrosis factor alpha) production, suggesting monocyte differentiation into inflammatory moDCs. The expansion of TNFalpha-producing CX3CR1(+) moDCs was associated with an elevation in hepatic and circulating TNFalpha level and with the worsening of parenchymal injury. Hydrogen sulfide (H2S) has been shown to interfere with CX3CR1 up-regulation in monocyte-derived cells exposed to pro-inflammatory stimuli. Treating 4-week-MCD-fed mice with the H2S donor NaHS while continuing on the same diet prevented the accumulation of TNFalpha-producing CX3CR1(+) moDCs without interfering with hepatic macrophage functions. Furthermore, NaHS reduced hepatic and circulating TNFalpha levels and ameliorated transaminase release and parenchymal injury. Altogether, these results show that inflammatory CX3CR1(+) moDCs contributed in sustaining inflammation and liver injury during steatohepatitis progression

Abstract: BACKGROUND AND AIM: Studies have verified the protective effect of
Hydrogen Sulfide (H2S) on gastric ulcer and ulcerative colitis, but the mechanisms are not fully illustrated. In this study, the possible protective effect of H2S on TNF-alpha/IFN-gamma induced barrier dysfunction was investigated in Caco-2 cell monolayers.

**METHOD:** The barrier function of Caco-2 monolayers was evaluated by measuring trans-epithelial electrical resistance (TEER) and FITC-Dextran 4 kDa (FD-4) trans-membrane flux. ZO-1 and Occludin were chosen as markers of the localization of tight junction (TJ) proteins for immunofluorescence. The expression of MLCK and phosphorylation level of myosin light chain (MLC) were measured by immunoblotting. The activation of NF-kB p65 was analyzed by EMSA and immunofluorescence.

**RESULTS:** NaHS at 500uM significantly attenuated TNF-alpha/IFN-gamma-induced Caco-2 monolayer barrier injury. The increased expression of MLCK and increased phosphorylation level of MLC induced by TNF-alpha/IFN-gamma was also inhibited significantly by NaHS. Additionally, NaHS inhibited TNF-alpha/IFN-gamma induced activation and nuclear translocation of NF-kB p65.

**CONCLUSION:** The present study reveals the protective effect of H2S on TNF-alpha and IFN-gamma-induced injury of intestinal epithelial barrier function in Caco-2 monolayers and suggests that the suppression of MLCK-P-MLC signaling mediated by NF-kB P65 might be one of the mechanisms underlying the protective effect of H2S.
C and chemical sequestration of N, Cl, and S by chromium result in quantitative conversion of compound-specific organic hydrogen to H2 analyte gas. The overall hydrogen isotope analysis via GC-Cr/HTC-isotope ratio mass spectrometry (IRMS) achieved a precision of better than +/- 5 mUr along the VSMOW-SLAP scale. The accuracy of GC-Cr/HTC-IRMS was validated with organic reference materials (RM) in comparison with online EA-Cr/HTC-IRMS and offline dual-inlet IRMS. The utility and reliability of the GC-Cr/HTC-IRMS system were documented during the routine measurement of more than 500 heteroatom-bearing organic samples spanning a delta(2)H range of -181 mUr to 629 mUr.


Abstract: The adsorption energy of reactant molecules and reaction intermediates is one of the key descriptors of catalytic activity of surfaces and is commonly used as a metric in screening materials for design of heterogeneous catalysts. The efficacy of such screening schemes depends on the accuracy of calculated adsorption energies under reaction conditions. These adsorption energies can depend strongly on interactions between adsorbed molecules in the adlayer. However, these interactions are typically not accounted for in screening procedures that use DFT-based zero-coverage adsorption energies. Identifying the physical mechanisms behind these interactions is essential to model realistic catalyst surfaces under reaction conditions and to understand the dependence of adsorption energies on reaction parameters like surface strain and composition. This article describes a method to quantitatively resolve the observed inter-adsorbate interactions into various direct adsorbate-adsorbate interactions (i.e. Coulombic and steric) and surface-mediated interactions (i.e. adsorbate-induced surface relaxation and change in electronic structure) by combining density functional theory and cluster-expansion calculations of coverage-dependent adsorption energies. The approach is implemented on a model catalyst surface of FeS2(100) reacting with H2S molecules. We find that the adsorption energy of H2S molecules can be affected by over 0.55 eV by the repulsive inter-adsorbate interactions caused primarily by the adsorbate-induced changes to the electronic structure of the FeS2 surface. These interactions also show a strong monotonic dependence on surface strain, being three times stronger on compressively strained surfaces than on surfaces under tensile strain. The large magnitude of inter-adsorbate interactions as well as their strong dependence on lattice strain demonstrate the need for using coverage-dependent adsorption energies for more accurate screening, for example for strained catalytic systems like core-shell and overlayer structures.


Abstract: A few-layered molybdenum disulfide (MoS2) thin film grown by plasma enhanced chemical vapor deposition was etched using a CF4 inductively coupled plasma, and the possibility of controlling the MoS2 layer thickness to a monolayer of MoS2 over a large area substrate was investigated. In addition, damage and contamination of the remaining MoS2 layer surface after etching and a possible method for film recovery was also investigated. The results from Raman spectroscopy and atomic force microscopy showed that one monolayer of MoS2 was etched by exposure to a CF4 plasma for 20 s after an initial incubation time of 20 s, i.e., the number of MoS2 layers could be controlled by exposure to the CF4 plasma for a certain processing time. However, XPS data showed that exposure to CF4 plasma induced a certain amount of damage and contamination by fluorine of the remaining MoS2 surface. After exposure to a H2S plasma for more than 10 min, the damage and fluorine contamination of the etched MoS2 surface could be effectively removed.
Abstract: Diallyl trisulfide (DATS) reacts rapidly with glutathione (GSH) to release H2S through thiol-disulfide exchange followed by allyl perthiol reduction by GSH. Yet diallyl disulfide (DADS) only releases a minute amount of H2S via a sluggish reaction with GSH through an alpha-carbon nucleophilic substitution pathway. The results clarify the misunderstanding of DADS as a rapid H2S donor, which is attributed to its DATS impurity.

Abstract: A superconductor is a material that can conduct electricity without resistance below a superconducting transition temperature, Tc. The highest Tc that has been achieved to date is in the copper oxide system: 133 Kelvin at ambient pressure and 164 Kelvin at high pressures. As the nature of superconductivity in these materials is still not fully understood (they are not conventional superconductors), the prospects for achieving still higher transition temperatures by this route are not clear. In contrast, the Bardeen-Cooper-Schrieffer theory of conventional superconductivity gives a guide for achieving high Tc with no theoretical upper bound—all that is needed is a favourable combination of high-frequency phonons, strong electron-phonon coupling, and a high density of states. These conditions can in principle be fulfilled for metallic hydrogen and covalent compounds dominated by hydrogen, as hydrogen atoms provide the necessary high-frequency phonon modes as well as the strong electron-phonon coupling. Numerous calculations support this idea and have predicted transition temperatures in the range 50-235 Kelvin for many hydrides, but only a moderate Tc of 17 Kelvin has been observed experimentally. Here we investigate sulfur hydride, where a Tc of 80 Kelvin has been predicted. We find that this system transforms to a metal at a pressure of approximately 90 gigapascals. On cooling, we see signatures of superconductivity: a sharp drop of the resistivity to zero and a decrease of the transition temperature with magnetic field, with magnetic susceptibility measurements confirming a Tc of 203 Kelvin. Moreover, a pronounced isotope shift of Tc in sulfur deuteride is suggestive of an electron-phonon mechanism of superconductivity that is consistent with the Bardeen-Cooper-Schrieffer scenario. We argue that the phase responsible for high-Tc superconductivity in this system is likely to be H3S, formed from H2S by decomposition under pressure. These findings raise hope for the prospects for achieving room-temperature superconductivity in other hydrogen-based materials.

Abstract: A dual signaling molecule sensor has received increasing attention owing to its ability to read out target analytes with more than one transduction channel and thus make the results more convincing. Here we have developed a dual signaling molecule sensor that is well suited for monitoring hydrogen sulfide (H2S) levels through fluorescence, UV-visible adsorption, and visual mode. Results showed the selective and instantaneous responses of sensor toward intracellular H2S. Moreover, the sensor was successfully applied to imaging of H2S levels in Caenorhabditis elegans (C. elegans) and observed the changes of H2S under starvation of C. elegans. Altogether, the sensor was proved to be a useful tool for tracking H2S levels in cells and in vivo. The merits of two kinds of independent signaling molecules allow us to select different output modes according to the different samples.

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Abstract: The reaction of a recently synthesized dihydroboron species complexed with bis(phosphinimino)amide, LBH2 (L = [N(Ph2PN(2,4,6-Me3C6H2))2]2(-)) with 3 equivalents of BH2Cl.SMe2 or one equivalent of BCl3 affords the first stable monohydridoborenium ion, [LBH]+[HBCl3](-) that is stable without a weakly coordinating bulky anion. Compound can also be prepared directly by refluxing LH with 3 equivalents of BH2Cl.SMe2. Interestingly, reaction of LBH2 (L) with elemental sulfur and selenium involves oxidative addition of S and Se into B-H bonds and subsequent release of H2S (or H2Se) from the intermediate LB(SH)2 (or LB(SeH)2) species forming stable compounds with terminal boron-chalcogen double bonds LB[double bond, length as m-dash]S () and LB[double bond, length as m-dash]Se (). The electronic structures of compounds, and were elucidated by high resolution mass spectrometry, multi-nuclear NMR and single crystal X-ray diffraction studies. Ab initio calculations on are in excellent agreement with its experimental structure and clearly support the existence of the boron-sulfur double bond.

Abstract: Atomic layer deposition (ALD) of cobalt sulfide (Co9S8) is reported. The deposition process uses bis(N,N'-diisopropylacetamidinato)cobalt(II) and H2S as the reactants and is able to produce high-quality Co9S8 films with an ideal layer-by-layer ALD growth behavior. The Co9S8 films can also be conformally deposited into deep narrow trenches with aspect ratio of 10:1, which demonstrates the high promise of this ALD process for conformally coating Co9S8 on high-aspect-ratio 3D nanostructures. As Co9S8 is a highly promising electrochemical active material for energy devices, we further explore its electrochemical performance by depositing Co9S8 on porous nickel foams for supercapacitor electrodes. Benefited from the merits of ALD for making high-quality uniform thin films, the ALD-prepared electrodes exhibit remarkable electrochemical performance, with high specific capacitance, great rate performance, and long-term cyclibility, which highlights the broad and promising applications of this ALD process for energy-related electrochemical devices, as well as for fabricating complex 3D nanodevices in general.

Abstract: This study describes a new method of passivating ZnO nanofiber-based devices with a ZnS layer. This one-step process was carried out in H2S gas at room temperature, and resulted in the formation of core/shell ZnO/ZnS nanofibers. This study presents the structural, optical and electrical properties of ZnO/ZnS nanofibers formed by a 2 nm ZnS sphalerite crystal shell covering a 5 nm ZnO wurtzite crystal core. The passivation process prevented free carriers from capture by oxygen molecules and significantly reduced the impact of O2 on nanostructure conductivity. The conductivity of the nanofibers was increased by three orders of magnitude after the sulfidation, the photoresponse time was reduced from 1500 s to 30 s, and the cathodoluminescence intensity increased with the sulfidation time thanks to the removal of ZnO surface defects by passivation. The ZnO/ZnS nanofibers were stable in water for over 30 days, and in phosphate buffers of acidic, neutral and alkaline pH for over 3 days. The by-products of the passivation process did not affect the conductivity of the devices. The potential of ZnO/ZnS nanofibers for protein biosensing is demonstrated using biotin and streptavidin as a model system. The presented ZnS shell preparation method can facilitate the construction of future sensors and protects the ZnO surface from dissolving in a biological environment.

Abstract: The separation of poisonous compounds from various process fluids has long been highly intractable, motivating the present study on the dynamic separation of H2S in acidic-gas-mixture-filled micropores. The molecular dynamics approach, coupled with the isothermal-isochoric ensemble, was used to model the molecular interactions and adsorption of H2S/CO2/CO/H2O mixtures inside metal-doped graphite slits. Due to the difference in the adsorption characteristics between the two distinct adsorbent materials, the metal dopant in the graphitic micropores leads to competitive adsorption, i.e. the Au and graphite walls compete to capture free adsorbates. The effects of competitive adsorption, coupled with changes in the gas temperature, concentration, constituent ratio and slit width on the constituent separation of mixtures were systematically studied. The molecule-wall binding energies calculated in this work (those of H2S, H2O and CO on Au walls and those of H2O, CO and CO2 on graphite walls) show good agreement with those obtained using density functional theory (DFT) and experimental results. The z-directional self-diffusivities (Dz) for adsorbates inside the slit ranged from 10(-9) to 10(-7) m(2) s(-1) as the temperature was increased from 10 to 500 K. The values are comparable with those for a typical microporous fluid (10(-8)-10(-9) m(2) s(-1) in a condensed phase and 10(-6)-10(-7) m(2) s(-1) in the gaseous state). The formation of H-bonding networks and hydrates of H2S is disadvantageous for the separation of mixtures. The results indicate that H2S can be efficiently separated from acidic gas mixtures onto the Au(111) surface by (i) reducing the mole fraction of H2S and H2O in the mixtures, (ii) raising the gas temperature to the high temperature limit (>/=400 K), and (iii) lowering the slit width to below the threshold dimension (</=23.26 A)

Abstract: Tetrathionate, a polythionate oxidation product of microbial hydrogen sulfide and reactive oxygen species from immune cells in the gut, serves as a terminal electron acceptor to confer a growth advantage for Salmonella and other enterobacteria. Here we show that the rat liver selenoenzyme thioredoxin reductase (Txnrd1, TR1) efficiently reduces tetrathionate in vitro. Furthermore, lysates of selenium-supplemented murine macrophages also displayed activity toward tetrathionate, while cells lacking TR1 were unable to reduce tetrathionate. These studies suggest that upregulation of TR1 expression, via selenium supplementation, may modulate the gut microbiome, particularly during inflammation, by regulating the levels of tetrathionate

Abstract: OPINION STATEMENT: Despite the introduction 20-30 years ago of potent inhibitors of gastric acid secretion and anti-inflammatory drugs that preferentially inhibit cyclo-oxygenase (COX)-2, the GI adverse effects of nonsteroidal anti-inflammatory drugs (NSAIDs) remain a significant clinical concern and a considerable economic burden. Inhibitors of acid secretion and selective COX-2 inhibitors reduce damage only in the proximal GI tract (stomach and proximal duodenum), but NSAIDs produce injury and bleeding throughout the GI tract. The small intestinal damage caused by NSAIDs is common, difficult to diagnose, and there are no proven-effective preventative or curative therapies. There is also emerging evidence that proton pump inhibitors (PPIs) and histamine H2-receptor antagonists (H2RAs) exacerbate NSAID-induced small intestinal injury. A new approach to solve this clinical problem is to deliver an endogenous, cytoprotective "rescue molecule" together with a COX inhibitor. Hydrogen sulfide (H2S) is a naturally produced, potent protective agent in the GI tract. H2S-releasing NSAIDs have been synthesized and extensively tested in laboratory animals and humans. They exhibit improved anti-inflammatory activity over the parent NSAID, while causing negligible damage in the GI tract.

Cortese-Krott MM, Kuhnle GG, Dyson A, Fernandez BO, Grman M, DuMond JF, et al. Key bioactive reaction products of the NO/H2S interaction are S/N-hybrid species, polysulfides, and nitroxy1. Proc Natl Acad Sci U S A 2015 Aug 25;112(34):E4651-E4660. Abstract: Experimental evidence suggests that nitric oxide (NO) and hydrogen sulfide (H2S) signaling pathways are intimately intertwined, with mutual attenuation or potentiation of biological responses in the cardiovascular system and elsewhere. The chemical basis of this interaction is elusive. Moreover, polysulfides recently emerged as potential mediators of H2S/sulfide signaling, but their biosynthesis and relationship to NO remain enigmatic. We sought to characterize the nature, chemical biology, and bioactivity of key reaction products formed in the NO/sulfide system. At physiological pH, we find that NO and sulfide form a network of cascading chemical reactions that generate radical intermediates as well as anionic and uncharged solutes, with accumulation of three major products: nitrosopersulfide (SSNO(-)), polysulfides, and dinitrososulfite [N-nitrosohydroxylamine-N-sulfonate (SULFI/NO)], each with a distinct chemical biology and in vitro and in vivo bioactivity. SSNO(-) is resistant to thiols and cyanolysis, efficiently donates both sulfane sulfur and NO, and potently lowers blood pressure. Polysulfides are both intermediates and products of SSNO(-) synthesis/decomposition, and they also decrease blood pressure and enhance arterial compliance. SULFI/NO is a weak combined NO/nitroxy1 donor that releases mainly N2O on decomposition; although it affects blood pressure only mildly, it markedly increases cardiac contractility, and formation of its precursor sulfite likely contributes to NO scavenging. Our results unveil an unexpectedly rich network of coupled chemical reactions between NO and H2S/sulfide, suggesting that the bioactivity of either transmitter is governed by concomitant formation of polysulfides and anionic S/N-hybrid species. This conceptual framework would seem to offer ample opportunities for the modulation of fundamental biological processes governed by redox switching and sulfur trafficking.

Liu MH, Lin XL, Zhang Y, He J, Tan TP, Wu SJ, et al. Hydrogen sulfide attenuates doxorubicin-induced cardiotoxicity by inhibiting reactive oxygen species-activated extracellular signal-regulated kinase 1/2 in H9c2 cardiac myocytes. Mol Med Rep 2015 Aug 21. Abstract: Doxorubicin (DOX) is a potent and available antitumor therapeutic agent; however, its clinical application is limited due to its cardiotoxicity. Preliminary evidence suggests that hydrogen sulfide (H2S) may exert protective effects on DOX-induced cardiotoxicity. Therefore, the aim of the present study was to investigate whether the extracellular signal-regulated kinase (ERK) 1/2 signaling pathway is involved in the cardioprotection of H2S against DOX-induced cardiotoxicity. The present study demonstrated that pretreatment with sodium hydrosulfide (NaHS; a donor of H2S) prior to DOX exposure attenuated the decreased cell viability, the increased apoptosis rate and the intracellular accumulation of reactive oxygen species (ROS) in H9c2 cardiac myocytes. Exposure of H9c2 cardiac myocytes to DOX upregulated the expression levels of phosphorylated ERK1/2, which had been reduced by pretreatment with NaHS or N-acetyl-L-cysteine, a ROS scavenger. In addition, H2S upregulated the antiapoptotic protein, Bcl2 and downregulated the proapoptotic protein, Bax. Notably, U0126, a selective inhibitor of ERK1/2, was observed to mimic the abovementioned cytoprotective activity of H2S. In conclusion, these findings indicate that H2S attenuates DOX-induced cardiotoxicity by inhibiting ROS-mediated activation of ERK1/2 in H9c2 cardiac myocytes.

the simultaneous hydrogen sulfide removal from biogas and nitrogen removal from swine slurry (Ssu-Nir) process. Anaerobic sludge, aerobic sludge, and water were used as inocula, and Na2S and biogas were used as a sulfide substrate, respectively. Additionally, 454 pyrosequencing of the 16S rRNA gene was used to explore the bacterial diversity. The results showed that sulfur-oxidizing bacteria (Thiobacillus, 42.2-84.4 %) were dominant in Ssu-Nir process and led to the excellent performance. Aerobic sludge was more suitable for inoculation of the Ssu-Nir process because it is better for rapidly enriching dominant sulfur-oxidizing bacteria (Thiobacillus, 54.4 %), denitrifying sulfur-oxidizing bacteria (40.0 %) and denitrifiers (23.9 %). Lower S2- removal efficiency (72.6 %) and

Abstract: Citrulline formation by both human neuronal nitric-oxide synthase (nNOS) and mouse macrophage inducible nitric-oxide synthase (iNOS) was inhibited by the hydrogen sulfide (H2S) donor Na2S with IC50-values of ~ 2.4x10-5 M and ~ 7.9x10-5 M, respectively, whereas human endothelial nitric-oxide synthase (eNOS) was hardly affected at all. Inhibition of nNOS was not affected by the concentrations of L-arginine (Arg), NADPH, FAD, FMN, tetrahydrobiopterin (BH4), and calmodulin (CaM), indicating that H2S does not interfere with substrate or cofactor binding. The IC50 decreased to ~ 1.5x10-5 M at pH 6.0 and increased to ~ 8.3x10-5 M at pH 8.0. Preincubation of concentrated nNOS with H2S under turnover conditions decreased activity after dilution by ~ 70 %, suggesting irreversible inhibition. However, when CaM was omitted during preincubation, activity was not affected, suggesting that irreversible inhibition requires both H2S and NO. Likewise, NADPH oxidation was inhibited with IC50 ~ 1.9x10-5 M in the presence of Arg and BH4, but exhibited much higher IC50-values (~ 1.0-6.1x10-4 M) when Arg and/or BH4 were omitted. Moreover, the relatively weak inhibition of nNOS by Na2S in the absence of Arg and/or BH4 was markedly potentiated by the NO-donor PROLI/NO (IC50 ~ 1.3-2.0x10-5 M). These results suggest that nNOS and iNOS, but not eNOS, are irreversibly inhibited by H2S/NO at modest concentrations of H2S in a reaction that may allow feedback inhibition of NO production under conditions of excessive NO/H2S formation

Abstract: Removal of hydrogen sulfide (H2S) and acid gases from natural gas is accomplished by absorption processes using a solvent. The gas solubility in a liquid can be used to measure the degree of removal of the gas and is quantified by the Henry's constant, the free energy of solvation at infinite dilution, or the excess chemical potential. In this work, Henry's constants and thermodynamic properties of solvation of H2S were calculated in three ionic liquids: [C4mim][PF6], [C4mim][BF4], and [C4mim][Cl] ([C4mim], 1-butyl-3-methyl imidazolium). The first step in this work was the evaluation of the force fields for the gas and condensed phases in order to obtain accurate values for the excess chemical potential for H2S on each ionic liquid using free energy perturbation techniques. In the H2S-[C4mim][PF6] and H2S-[C4mim][BF4] systems, the results obtained by molecular simulation agree with the experimental values reported in the literature. However, the solvation free energy calculated for the H2S-[C4mim][Cl] system can be considered predictive because of the lack of experimental data at the simulated conditions. Based on these results, the best solvent for removing H2S is [C4mim][Cl] because it has the highest affinity for this species (lowest value of the Henry's constant). Also, solvation thermodynamic properties such as enthalpy and entropy were calculated in order to evaluate their contribution to the free energy of solvation
Abstract: Hydrogen sulfide (H2S) is the third gaseous signaling molecule that plays important roles in cancer biological processes. Recent studies indicate that H2S has both pro-cancer and anti-cancer effects. Endogenous H2S can exert pro-cancer functions through induction of angiogenesis regulation of mitochondrial bioenergetics, acceleration of cell cycle progression, and anti-apoptosis mechanisms. Thus, the inhibition of the production of H2S in cancer cells may be a new cancer treatment strategy. In contrast to the pro-cancer effect of H2S, relatively high concentrations of exogenous H2S could suppress the growth of cancer cells by inducing uncontrolled intracellular acidification, inducing cell cycle arrest, and promoting apoptosis. Therefore, H2S donors and H2S-releasing hybrids could be designed and developed as novel anti-cancer drugs. In this review, the production and metabolism of H2S in cancer cells are summarized and the role and mechanism of H2S in cancer development and progression are further discussed.

Abstract: The room temperature chemiresistive response of n-type ZnO nanowire (ZnO NWs) films modified with different thicknesses of p-type cobalt phthalocyanine (CoPc) has been studied. With increasing thickness of CoPc (>15 nm), heterojunction films exhibit a transition from n- to p-type conduction due to uniform coating of CoPc on ZnO. The heterojunction films prepared with a 25 nm thick CoPc layer exhibit the highest response (268% at 10 ppm of H2S) and the fastest response (26 s) among all samples. The X-ray photoelectron spectroscopy and work function measurements reveal that electron transfer takes place from ZnO to CoPc, resulting in formation of a p-n junction with a barrier height of 0.4 eV and a depletion layer width of approximately 8.9 nm. The detailed XPS analysis suggests that these heterojunction films with 25 nm thick CoPc exhibit the least content of chemisorbed oxygen, enabling the direct interaction of H2S with the CoPc molecule, and therefore exhibit the fastest response. The improved response is attributed to the high susceptibility of the p-n junctions to the H2S gas, which manipulates the depletion layer width and controls the charge transport.

Abstract: BACKGROUND: Both hydrogen sulphide (H2S) and mild hypothermia have been reported to prevent brain damage caused by reperfusion assault through regulating the N-methyl-D-aspartate receptor (NMDAR). However, the relationship between the two treatments and how they exert neuro-protective effects through NMDARs remain to be elucidated. METHODS: Transient cerebral ischemia was induced using the Pulsinelli four-vessel occlusion method. We used sodium hydrosulphide (NaHS) as the H2S donor. We randomly divided 100 Sprague-Dawley rats into five groups of 20: Sham operation group (Sh), normothermic (36-37 degrees C) ischemia group (NT), mild hypothermic (32-33 degrees C) ischemia group (mHT), normothermic ischemia combined with NaHS treatment group (NT + NaHS), and mild hypothermic ischemia combined with NaHS treatment group (mHT + NaHS). After 6 hrs of reperfusion, rats were decapitated and hippocampus samples were immediately collected. We measured NR2A (GluN1), NR2B (GluN2) and p-CREB protein levels using western blotting. We further analyzed BDNF mRNA expression by real-time PCR. Hematoxylin and eosin (HE) staining was used to examine pyramidal cell histology at the CA1 region. All statistical analyses were carried out by ANOVA and LSD t-test as implemented by the SPSS 13.0 software. RESULTS: In the four test groups with ischemia-reperfusion, hippocampal H2S concentration increased following treatment, and administration of NaHS further increased H2S levels. Moreover, administration of both NaHS and mild hypothermia resulted in up-regulation of NR2A and
NR2B protein expressions, as well as p-CREB protein and BDNF mRNA levels. At the cellular level, NaHS and mild hypothermia groups exhibited lower damage caused by ischemia-reperfusion in the CA1 region of the hippocampus. The strongest protective effect was observed in rats treated with combined NaHS and mild hypothermia, suggesting their effects were additive. CONCLUSION: Our results support previous findings that hydrogen sulphide and mild hypothermia can prevent ischemia-reperfusion injury. Both treatments caused an up-regulation of NMDA receptors, as well as an elevation in p-CREB protein and BDNF mRNA levels. Thus, hydrogen sulphide and mild hypothermia may provide neuro-protective effect through activating the pro-survival CREB signaling pathway.

Abstract: Demethylation of the Foxp3 locus maintains gene expression and Treg cell stability. Yang et al. (2015) show that the gasotransmitter hydrogen sulfide co-operates with growth factor TGF-beta and interleukin-2 to activate Tet-mediated DNA demethylation of Foxp3 to promote immune tolerance.

Abstract: Regulatory T (Treg) cells are essential for maintenance of immune homeostasis. Here we found that hydrogen sulfide (H2S) was required for Foxp3(+)- Treg cell differentiation and function and that H2S deficiency led to systemic autoimmune disease. H2S maintained expression of methylcytosine dioxygenases Tet1 and Tet2 by sulfhydrating nuclear transcription factor Y subunit beta (NFYB) to facilitate its binding to Tet1 and Tet2 promoters. Transforming growth factor-beta (TGF-beta)-activated Smad3 and interleukin-2 (IL-2)-activated Stat5 facilitated Tet1 and Tet2 binding to Foxp3. Tet1 and Tet2 catalyzed conversion of 5-methylcytosine (5mC) to 5-hydroxymethylcytosine (5hmC) in Foxp3 to establish a Treg-cell-specific hypomethylation pattern and stable Foxp3 expression. Consequently, Tet1 and Tet2 deletion led to Foxp3 hypermethylation, impaired Treg cell differentiation and function, and autoimmune disease. Thus, H2S promotes Tet1 and Tet2 expression, which are recruited to Foxp3 by TGF-beta and IL-2 signaling to maintain Foxp3 demethylation and Treg-cell-associated immune homeostasis.

Abstract: In addition to its role in the endogenous synthesis of cysteine, cystathionine gamma-lyase (CGL) is a major physiological source of the vasorelaxant hydrogen sulfide. Cgl null mice are potentially useful for studying the influence of this compound upon vascular tone and endothelial function. Here, we confirm a previous report that female Cgl null mice exhibit an approximate 45-fold increase in plasma total homocysteine compared to wild type controls. This level of homocysteine is approximately 3.5-fold higher than that observed in male Cgl null mice and is essentially equivalent to that observed in mouse models of cystathionine beta synthase deficient homocystinuria. Cgl null mice of both sexes exhibited decreased expression of methylenetetrahydrofolate reductase and cysteinesulfinate decarboxylase compared to WT controls. Female Cgl null mice exhibited a sex-specific induction of betaine homocysteine S-methyltransferase and methionine adenosyltransferase 1, alpha and a 70% decrease in methionine synthase expression accompanied by significantly decreased plasma methionine. Decreased plasma cysteine levels in female Cgl null mice were associated with sex-specific dysregulation of cysteine dioxygenase expression. Comparative histological assessment between cystathionine beta-synthase and Cgl null mice indicated that the
therapeutic potential of cystathionine against liver injury merits possible further investigation. Collectively, our data demonstrates the importance of considering sex when investigating mouse models of inborn errors of metabolism and indicate that while female Cgl null mice are of questionable utility for studying the physiological role of hydrogen sulfide, they could serve as a useful model for studying the consequences of methionine synthase deficiency and the methylfolate trap.


Abstract: Sulindac is chemopreventive and has utility in patients with familial adenomatous polyposis; however, side effects preclude its long-term use. NOSH-sulindac (AVT-18A) releases nitric oxide and hydrogen sulfide, was designed to be a safer alternative. Here we compare the gastrointestinal safety, anti-inflammatory, analgesic, anti-pyretic, anti-platelet, and anti-cancer properties of sulindac and NOSH-sulindac administered orally to rats at equimolar doses. Gastrointestinal safety: 6h post-administration, number/size of hemorrhagic lesions in stomachs were counted. Tissue samples were frozen for PGE2, SOD, and MDA determination. Anti-inflammatory: 1h after drug administration, the volume of carrageenan-induced rat paw edemas was measured for 5h. Anti-pyretic: fever was induced by LPS (ip) an hour before administration of the test drugs, core body temperature was measured hourly for 5h. Analgesic: time-dependent analgesic effects were evaluated by carrageenan-induced hyperalgesia. Antiplatelet: anti-aggregatory effects were studied on collagen-induced platelet aggregation of human platelet-rich plasma. Anti-cancer: We examined the effects of NOSH-sulindac on the growth properties of 12 human cancer cell lines of six different tissue origins. Both agents reduced PGE2 levels in stomach tissue; however, NOSH-sulindac did not cause any stomach ulcers, whereas sulindac caused significant bleeding. Lipid peroxidation induced by sulindac was higher than that from NOSH-sulindac. SOD activity was significantly lowered by sulindac but increased by NOSH-sulindac. Both agents showed similar anti-inflammatory, analgesic, anti-pyretic, and anti-platelet activities. Sulindac increased plasma TNFalpha whereas this rise was lower in the NOSH-sulindac-treated animals. NOSH-sulindac inhibited the growth of all cancer cell lines studied, with potencies of


Abstract: One of the problems in waste water treatment plants (WWTPs) is the increase in emissions of hydrogen sulfide (H2S), which can cause damage to the health of human populations and ecosystems. To control emissions of this gas, sulfur-oxidizing bacteria can be used to convert H2S to sulfate. In this work, sulfate detection was performed by spectrophotometry, ion chromatography and atomic absorption spectrometry, using Paracoccus pantotrophus ATCC 35512 as a reference strain growing in inorganic broth supplemented with sodium thiosulfate (Na2S2O3.5H2O), sodium sulfide (Na2S) or sodium sulfite (Na2SO3), separately. The strain was metabolically competent in sulfate production. However, it was only possible to observe significant differences in sulfate production compared to abiotic control when the inorganic medium was supplemented with sodium thiosulfate. The three methods for sulfate detection showed similar patterns, although the chromatographic method was the most sensitive for this study. This strain can be used as a reference for sulfate production in studies with sulfur oxidizing bacteria originating from environmental samples of WWTPs

Abstract: BACKGROUND: Many of the invading oral bacteria are known to produce considerable amounts of hydrogen sulfide (H2S). The toxic activity of exogenous H2S in periodontal tissue has been demonstrated, however, the role of endogenous H2S in the physiological function of periodontal tissue remains poorly understood. The purpose of the present study was to investigate the biological functions of hydrogen sulfide (H2S) on the proliferation and differentiation of human periodontal ligament stem cells (PDLSCs).

METHODS: PDLSCs were isolated from the periodontal ligament tissues of normal human volunteers or patients with periodontitis. Immunocytochemical staining, flow cytometry and western blot were used to examine the expression of H2S synthesizing enzymes CBS and CSE. The proliferation capacity of PDLSCs was determined by CCK-8 assay, CFSE analysis, and EdU assay. The osteogenic potential of PDLSCs was tested using ALP staining, alizarin red staining, and in vivo transplantation experiments. Oil red staining was used to analyze the adipogenic ability. RESULTS: We find that human PDLSCs express both CBS and CSE and produce H2S. Blocking the generation of endogenous H2S with CBS inhibitor hydroxylamine (HA) significantly attenuated PDLSCs proliferation and reduced the osteogenic and adipogenic differentiation capacity of PDLSCs. In contrast, CSE inhibitor DL-propargylglycine (PAG) had no effect on PDLSCs function. Exogenous H2S could inhibit the production of endogenous H2S and impair PDLSCs function in a dose-dependent manner. CONCLUSIONS: The physiological level of endogenous H2S maintains the proliferation and differentiation capacity of PDLSCs, and CBS may be the main source of endogenous H2S in PDLSCs.


Abstract: Rhodanese is a component of the mitochondrial hydrogen sulfide (H2S) oxidation pathway. Rhodanese catalyzes the transfer of sulfane sulfur from glutathione persulfide (GSSH) to sulfite generating thiosulfate and from thiosulfate to cyanide generating thiocyanate. Two polymorphic variations have been identified in the rhodanese coding sequence in the French Caucasian population. The first, 306A-->C, has an allelic frequency of 1 percent and results in an E102D substitution in the encoded protein. The second polymorphism 853C-->G, has an allelic frequency of 5 percent and leads to a P285A substitution. In this study, we have examined differences in the stability between wild-type rhodanese and the E102D and P285A variants and in the kinetics of the sulfurtransfer reactions. The Asp102 and Ala285 variants are more stable than wild-type rhodanese and exhibit kcat/KM,CN values that are 17 and 1.6 fold higher, respectively. All three rhodanese forms preferentially catalyze sulfurtransfer from GSSH to sulfite, generating thiosulfate and glutathione. The kcat/KM,sulfite values for the variants in the sulfurtransfer reaction from GSSH to sulfite were 1.6 (Asp102) and 4 fold (Ala285) lower than for wild-type rhodanese while the kcat/KM,GSSH values were similar for all three enzymes. Thiosulfate-dependent H2S production in murine liver lysate is low, consistent with a role for rhodanese in sulfide oxidation. Our studies show that polymorphic variations that are distant from the active site differentially modulate the sulfurtransferase activity of human rhodanese to cyanide versus sulfite and might be important in differences in susceptibility to diseases where rhodanese dysfunction has been implicated, e.g. inflammatory bowel diseases.


Abstract: Hydrogen sulfide (H2S) is increasingly recognized to modulate physiological processes in mammals through mechanisms that are currently under scrutiny. H2S is not able to react with reduced thiols (RSH). However, H2S -precisely, HS-- is able to react with oxidized thiol derivatives. We performed a systematic study of the reactivity of HS-toward symmetric low molecular weight disulfides (RSSR) and mixed albumin (HSA) disulfides. Correlations with thiol acidity and computational modeling showed that the reaction occurs through a concerted mechanism. Comparison to analogous reactions of
thiolates indicated that the intrinsic reactivity of HS- is one order of magnitude lower than that of thiolates. In addition, H2S is able to react with sulfenic acids (RSOH). The rate constant of the reaction of H2S with the sulfenic acid formed in HSA was determined. Both reactions of H2S with disulfides and sulfenic acids yield persulfides (RSSH), recently identified post-translational modifications. The formation of this derivative in HSA was determined and the rate constants of its reactions with a reporter disulfide and with peroxynitrite revealed that persulfides are better nucleophiles than thiols, consistent with the alpha effect. Experiments with cells in culture showed that treatment with hydrogen peroxide enhanced the formation of persulfides. Biological implications are discussed. Our results give light on the mechanisms of persulfide formation and provide quantitative evidence for the high nucleophilicity of these novel derivatives, setting the stage for understanding the contribution of the reactions of H2S with oxidized thiol derivatives to H2S effector processes.

(35) Ju Y, Untereiner A, Wu L, Yang G. HS-induced S-sulfhydration of pyruvate carboxylase contributes to gluconeogenesis in liver cells. Biochim Biophys Acta 2015 Aug 11;1850(11):2293-303. Abstract: BACKGROUND: Cystathionine gamma-lyase (CSE)-derived hydrogen sulfide (H2S) possesses diverse roles in the liver, affecting lipoprotein synthesis, insulin sensitivity, and mitochondrial biogenesis. H2S S-sulfhydration is now proposed as a major mechanism for H2S-mediated signaling. Pyruvate carboxylase (PC) is an important enzyme for gluconeogenesis. S-sulfhydration regulation of PC by H2S and its implication in gluconeogenesis in the liver have been unknown. METHODS: Gene expressions were analyzed by real-time PCR and western blotting, and protein S-sulfhydration was assessed by both modified biotin switch assay and tag switch assay. Glucose production and PC activity was measured with coupled enzyme assays, respectively. RESULTS: Exogenously applied H2S stimulates PC activity and gluconeogenesis in both HepG2 cells and mouse primary liver cells. CSE overexpression enhanced but CSE knockout reduced PC activity and gluconeogenesis in liver cells, and blockage of PC activity abolished H2S-induced gluconeogenesis. H2S had no effect on the expressions of PC mRNA and protein, while H2S S-sulfhydrated PC in a dithiothreitol-sensitive way. PC S-sulfhydration was significantly strengthened by CSE overexpression but attenuated by CSE knockout, suggesting that H2S enhances glucose production through S-sulfhydrating PC. Mutation of cysteine 265 in human PC diminished H2S-induced PC S-sulfhydration and activity. In addition, high-fat diet feeding of mice decreased both CSE expression and PC S-sulfhydration in the liver, while glucose deprivation of HepG2 cells stimulated CSE expression. CONCLUSIONS: CSE/H2S pathway plays an important role in the regulation of glucose production through S-sulfhydrating PC in the liver. GENERAL SIGNIFICANCE: Tissue-specific regulation of CSE/H2S pathway might be a promising therapeutic target of diabetes and other metabolic syndromes.

(36) Syhr KM, Boosen M, Hohmann SW, Longen S, Kohler Y, Pfeilschifter J, et al. The HS-producing enzyme CSE is dispensable for the processing of inflammatory and neuropathic pain. Brain Res 2015 Aug 10. Abstract: Accumulating lines of evidence indicate that hydrogen sulfide (H2S) contributes to the processing of chronic pain. However, the sources of H2S production in the nociceptive system are poorly understood. Here we investigated the expression of the H2S releasing enzyme cystathionine gamma-lyase (CSE) in the nociceptive system and characterized its role in chronic pain signaling using CSE deficient mice. We show that paw inflammation and peripheral nerve injury led to upregulation of CSE expression in dorsal root ganglia. However, conditional knockout mice lacking CSE in sensory neurons as well as global CSE knockout mice demonstrated normal pain behaviors in inflammatory and neuropathic pain models as compared to WT littermates. Thus, our results suggest that CSE is not critically involved in chronic pain signaling in mice and that sources different from CSE mediate the pain relevant effects of H2S.
Auguet O, Pijuan M, Batista J, Borrego CM, Gutierrez O. Changes of microbial biofilm communities during colonization of sewer systems. Appl Environ Microbiol 2015 Aug 7. Abstract: Coexistence of sulfate-reducing bacteria (SRB) and methanogenic archaea (MA) in anaerobic biofilms developed in sewer inner pipe surfaces favours the accumulation of sulfide (H2S) and methane (CH4) as metabolic end products, causing severe impacts on sewerage systems. In this study we investigated the time-course of H2S and CH4 production and emission rates during different stages of biofilm development in relation to changes in the composition of microbial biofilm communities. The study was carried out in a laboratory sewer pilot plant that mimics a full-scale anaerobic rising sewer using a combination of process data and molecular techniques (e.g. qPCR, DGGE and 16S rRNA gene pyrotag sequencing). After two weeks of biofilm growth, H2S emission was notably high (290.7 +/- 72.3 mg S-H2S l-1 day-1) whereas emissions of CH4 remained low (17.9 +/- 15.9 mg COD-CH4 l-1 day-1). This contrasting trend coincided with a stable SRB community and an archaeal community solely composed of methanogens derived from the human gut (i.e. Methanobrevibacter and Methanosphaera). In turn, CH4 emissions increased after one year of biofilm growth (327.6 +/- 16.6 mg COD-CH4 l-1 day-1) coinciding with the replacement of methanogenic colonizers by species more adapted to sewer conditions (i.e. Methanosaeta spp.). Our study provides data that confirm the capacity of our laboratory experimental system to mimic the functioning of full-scale sewers both microbiologically and operationally in terms of sulfide and methane production, gaining insight on the complex dynamics of key microbial groups during biofilm development.

Fukami K, Sekiguchi F, Yasukawa M, Asano E, Kasamatsu R, Ueda M, et al. Functional upregulation of the HS/Ca3.2 channel pathway accelerates secretory function in neuroendocrine-differentiated human prostate cancer cells. Biochem Pharmacol 2015 Aug 7. Abstract: Neuroendocrine-differentiated prostate cancer cells may contribute to androgen-independent proliferation of surrounding cells through Ca2+-dependent secretion of mitogenic factors. Human prostate cancer LNCaP cells, when neuroendocrine-differentiated, overexpress Cav3.2 T-type Ca2+ channels that contribute to Ca2+-dependent secretion. Given evidence for the acceleration of Cav3.2 activity by hydrogen sulfide (H2S), we examined the roles of the H2S/Cav3.2 pathway and then analyzed the molecular mechanisms of the Cav3.2 overexpression in neuroendocrine-differentiated LNCaP cells. LNCaP cells were differentiated by dibutyryl cyclic AMP. Protein levels and T-type Ca2+ channel-dependent currents (T-currents) were measured by immunoblotting and whole-cell patch-clamp technique, respectively. Spontaneous release of prostatic acid phosphatase (PAP) was monitored to evaluate secretory function. The differentiated LNCaP cells exhibited neurite outgrowth, androgen-independent proliferation and upregulation of mitogenic factors, and also showed elevation of Cav3.2 expression or T-currents. Expression of cystathionine-gamma-lyase (CSE) and cystathionine-beta-synthase (CBS), H2S-forming enzymes, and spontaneous secretion of PAP increased following the differentiation. The augmented T-currents were enhanced by H2S donors and suppressed by inhibitors of CSE, but not CBS. The PAP secretion was reduced by inhibition of CSE or T-type Ca2+ channels. During differentiation, Egr-1 and REST, positive and negative transcriptional regulators for Cav3.2, were upregulated and downregulated, respectively, and Egr-1 knockdown prevented the Cav3.2 overexpression. Our data suggest that, in neuroendocrine-differentiated LNCaP cells, H2S formed by the upregulated CSE promotes the activity of the upregulated Cav3.2, leading to the elevated secretory functions. The overexpression of Cav3.2 appears to involve upregulation of Egr-1 and downregulation of REST.

it plays an important role in various physiological processes and pathological processes in vivo, such as vasodilation, apoptosis, neurotransmission, ischemia/reperfusion-induced injury, insulin secretion and inflammation. Developing a highly selective and sensitive method that can detect H2S in the biological system is very important. In this work, a colorimetric and "turn-on" fluorescent probe is developed. Furthermore, this probe displays a highly selective response to H2S in aqueous solution and possesses good capability for bioimaging H2S without interference in living cells. The results suggest that a H2S-selective probe has good water-solubility, biocompatibility and cell-penetrability and can serve as an efficient tool for probing H2S in the cell level.

(40) Abbott T, Eskicioglu C. Effects of metal salt addition on odor and process stability during the anaerobic digestion of municipal waste sludge. Waste Manag 2015 Aug 7. Abstract: Anaerobic digestion (AD) is an effective way to recover energy and nutrients from organic waste; however, several issues including the solubilization of bound nutrients and the production of corrosive, highly odorous and toxic volatile sulfur compounds (VSCs) in AD biogas can limit its wider adoption. This study explored the effects of adding two different doses of ferric chloride, aluminum sulfate and magnesium hydroxide directly to the feed of complete mix semi-continuously fed mesophilic ADs on eight of the most odorous VSCs in AD biogas at three different organic loading rates (OLR). Ferric chloride was shown to be extremely effective in reducing VSCs by up to 87%, aluminum sulfate had the opposite effect and increased VSC levels by up to 920%, while magnesium hydroxide was not shown to have any significant impact. Ferric chloride, aluminum sulfate and magnesium hydroxide were effective in reducing the concentration of orthophosphate in AD effluent although both levels of alum addition caused digester failure at elevated OLRS. Extensive foaming was observed within the magnesium hydroxide dosed digesters, particularly at higher doses and high OLRS. Certain metal salt additions may be a valuable tool in overcoming barriers to AD and to meet regulatory targets.

(41) Schachtli E, Kondratieva E, Gutierrez OY, Lercher JA. Pathways for H2 Activation on (Ni)-MoS2 Catalysts. J Phys Chem Lett 2015 Aug 6;6(15):2929-32. Abstract: The activation of H2 and H2S on (Ni)MoS2/Al2O3 leads to the formation of SH groups with acid character able to protonate 2,6-dimethylpyridine. The variation in concentrations of SH groups induced by H2 and H2S adsorption shows that both molecules dissociate on coordinatively unsaturated cations and neighboring S(2-). In the studied materials, one sulfur vacancy and four SH groups per 10 metal atoms exist at the active edges of MoS2 under the conditions studied. H2-D2 exchange studies show that Ni increases the concentration of active surface hydrogen by up to 30% at the optimum Ni loading, by increasing the concentration of H2 and H2S chemisorption sites.

(42) Chiumenti A. Complete nitrification-denitrification of swine manure in a full-scale, non-conventional composting system. Waste Manag 2015 Aug 6. Abstract: A full-scale composting plant (track type, aerated by screws), treating liquid swine manure (94.8% on mass basis) with straw (<0.8%) and sawdust (4.4%), was monitored. The main objectives were testing the performance of the process and assessing its environmental sustainability. Particular attention was dedicated to verify the possibility that this process could determine significant mass reduction, along with Nitrogen reduction, mainly by denitrification. Emissions were evaluated by measuring NH3, N2O and CH4 (by static chamber), H2S and odor emissions (by dynamic olfactometry). Quality and quantity of inputs and outputs and process parameters (redox, oxygen, and temperature) were monitored. The process produced a mature, highly humified (Humification Index=0.27), solid product with 92.8% mass reduction (mainly evaporation), and nitrogen reduction (85.8% referred to input TN). The process was revealed to be environmentally sustainable: emissions of odors and H2S resulted negligible; emissions of N-N2O represented 0.18% of TN input, while emissions of N-NH3 represented 0.87% of input TN. Microbiological analyses determined the
presence of 107 CFU/g of bacteria related to N cycle and real time PCR demonstrated the presence in the final product of 4.77×10⁷ couples of genes of Bacterial amoA/gTS and 2.46×10⁷ couples NosZ/gTS, indicating nitrification and complete denitrification. These results exhibit that nitrification and complete denitrification can efficiently occur in a composting process effectively transforming N₂O into N₂ as consequence of the optimized alternation of aerated and anoxic phases in the feedstock.


Abstract: In heart disease, transforming growth factor-beta1 (TGF-beta1) converts fibroblasts into myofibroblasts, which synthesize and secrete fibrillar type I and III collagens. The purpose of the present study was to investigate how hydrogen sulfide (H₂S) suppresses TGF-beta1-induced differentiation of human cardiac fibroblasts to myofibroblasts. Human cardiac fibroblasts were serum-starved in fibroblast medium for 16 h before exposure to TGF-beta1 (10 ng mL⁻¹) for 24 h with or without sodium hydrosulfide (NaHS, 100 μmol L⁻¹, 30 min pretreatment) treatment. NaHS, an exogenous H₂S donor, potently inhibited the proliferation and migration of TGF-beta1-induced human cardiac fibroblasts and regulated their cell cycle progression. Furthermore, NaHS treatment led to suppression of fibroblast differentiation into myofibroblasts, and reduced the levels of collagen, TGF-beta1, and activated Smad3 in TGF-beta1-induced human cardiac fibroblasts in vitro. We therefore conclude that H₂S suppresses TGF-beta1-stimulated conversion of fibroblasts to myofibroblasts by inhibiting the TGF-beta1/Smad3 signaling pathway, as well as by inhibiting the proliferation, migration, and cell cycle progression of human cardiac myofibroblasts. These effects of H₂S may play significant roles in cardiac remodeling associated with heart failure.


Abstract: AIMS: Myocardial infarction followed by adverse left ventricular (LV) remodeling is the most frequent proximate cause of heart failure. Hydrogen sulfide (H₂S) is an important endogenous modulator of diverse physiological and pathophysiological processes. Its role in post-ischemic ventricular remodeling and the associated neurohormonal responses has not been defined. Here, we aimed at evaluating whether the slow-releasing water-soluble H₂S donor GYY4137 (GYY) exerts cardioprotective effects and modulates the neurohormonal response to cardiac ischemic injury.

METHODS AND RESULTS: Treatment for 2 or 7 days with GYY (100 mg/Kg/48h, IP) after acute myocardial infarction (MI) in rats preserved LV dimensions and function in vivo, compared to untreated infarcted (MI), placebo- and dl-propargylglycine- (PAG, an inhibitor of endogenous H₂S synthesis) treated animals (n=9/group/time-point). LV dimensions and function in GYY-treated animals were comparable to healthy sham-operated rats. GYY-treated hearts had significantly less LV fibrosis than MI, placebo and PAG hearts. A higher density of blood vessels was found in the LV scar area of GYY-treated animals compared to all other infarcted groups. Despite preserved LV structure and function, treatment with GYY increased the levels of the natriuretic peptides ANP and BNP in association with enhanced cyclic GMP levels, paralleled by higher cGMP-dependent protein kinase type I (cGKII) protein levels. CONCLUSIONS: Our data suggest that the slow-releasing H₂S donor, GYY4137, preserves cardiac function, attenuates adverse remodeling and may exert post-ischemic cardioprotective (pro-angiogenic, anti-apoptotic, anti-hypertrophic and anti-fibrotic) effects in part through enhanced early post-ischemic endogenous natriuretic peptide activation.

Abstract: Odour pollution caused by municipal solid waste is a public concern. This study quantitatively evaluated the concentration, environmental impacts, and olfaction of volatile trace compounds released from a waste transfer station. Seventy-six compounds were detected, and ethanol presented the highest releasing rate and ratio of 14.76kg/d and 12.30g/t of waste, respectively. Life cycle assessment showed that trichlorofluoromethane and dichlorodifluoromethane accounted for more than 99% of impact potentials to global warming and approximately 70% to human toxicity (non-carcinogenic). The major contributor for both photochemical ozone formation and ecotoxicity was ethanol. A detection threshold method was also used to evaluate odour pollution. Five compounds including methane thiol, hydrogen sulphide, ethanol, dimethyl disulphide, and dimethyl sulphide, with dilution multiples above one, were considered the critical compounds. Methane thiol showed the highest contribution to odour pollution of more than 90%, as indicated by its low threshold. Comparison of the contributions of the compounds to different environmental aspects indicated that typical pollutants varied based on specific evaluation targets and therefore should be comprehensively considered. This study provides important information and scientific methodology to elucidate the impacts of odourant compounds to the environment and odour pollution.


Abstract: The main components of oral malodor have been identified as volatile sulfur compounds (VSCs) including hydrogen sulfide and methyl mercaptan. The lactoperoxidase (LPO) system (consisting of LPO, glucose oxidase, glucose, and thiocyanate) has been reported to exhibit antimicrobial activities against oral bacteria in vitro and suppressive effects on VSCs in mouth air in a clinical trial. We herein examined the in vitro effects of the LPO system on the activities of the bacterial lyases involved in the production of VSCs by oral anaerobes. The exposure of crude bacterial extracts of Fusobacterium nucleatum and Porphyromonas gingivalis or purified methionine gamma-lyase to the LPO system resulted in the inactivation of their lyase activities through L-cysteine and L-methionine, which was linked to the production of hydrogen sulfide and methyl mercaptan, respectively. Exposing living F. nucleatum and P. gingivalis cells to the LPO system resulted in the suppression of cell numbers and lyase activities. The inactivation of the crude bacterial extracts of F. nucleatum and purified methionine gamma-lyase by the LPO system was partly recovered by the addition of dithiothreitol. Therefore, the LPO system may inactivate bacterial lyases including methionine gamma-lyase by reacting with the free cysteine residues of lyases. These results suggest that the LPO system suppresses the production of VSCs not only through its antimicrobial effects, but also by its inactivating effects on the bacterial lyases of VSC-producing bacteria.


Abstract: PURPOSE: Ischemia-reperfusion injury is unavoidable during organ transplantation. Prolonged ischemia-reperfusion injury is detrimental to short-term and long-term graft function and survival. H2S is a recently characterized, endogenously produced gaseous molecule with important physiological roles that has been shown to be cytoprotective during tissue ischemia-reperfusion injury. The current study aimed to determine whether H2S could mitigate cold renal ischemia-reperfusion injury in the clinically relevant context of allogeneic renal transplantation. MATERIALS AND METHODS: Following bilateral native nephrectomy Lewis rats underwent renal transplantation with kidneys from Brown Norway donor rats that were flushed with cold (4°C) standard University of Wisconsin preservation solution (University of Wisconsin preservation solution group) or cold University of Wisconsin preservation solution plus
150 μM NaHS (H2S group) solution. Kidneys were stored for 6 hours at 4°C in the same solution. Recipient animals were monitored for 14 days or until sacrifice using metabolic cages to assess various parameters of renal graft function. RESULTS: H2S treatment improved early allograft survival and function, and decreased early levels of necrosis, apoptosis and Kim-1 compared to University of Wisconsin preservation solution alone. H2S treatment did not affect allograft rejection. Rather, it modulated the early allograft transcriptome to decrease the expression of renal injury, coagulation and cellular stress response genes, and increase the expression of cellular proliferation and Ifn-gamma induced genes compared to University of Wisconsin preservation solution alone. CONCLUSIONS: To our knowledge our findings are the first to show that H2S protects donor kidneys against cold ischemia-reperfusion injury in the context of allogeneic renal transplantation. This potentially represents a novel cost-effective therapeutic solution to mitigate ischemia-reperfusion injury and improve the clinical outcomes of renal transplantation

Abstract: Compelling evidence suggests that hydrogen sulfide represents an important gaseous transmitter in the mammalian respiratory system. In the present study, we have evaluated the role of mast cells in hydrogen sulfide-induced effects on airways in a mouse model of asthma. Mice were sensitized to ovalbumin and received aerosol of a hydrogen sulfide donor (NaHS; 100ppm) starting at day 7 after ovalbumin challenge. Exposure to hydrogen sulfide abrogated ovalbumin-induced bronchial hyperreactivity as well as the increase in lung resistance. Concomitantly, hydrogen sulfide prevented mast cell activity as well as FGF-2 and IL-13 upregulation. Conversely, pulmonary inflammation and the increase in plasmatic IgE levels were not affected by hydrogen sulfide. A lack of hydrogen sulfide effects in mast cell deficient mice occurred. Primary fibroblasts harvested from ovalbumin-sensitized mice showed an increased proliferation rate that was inhibited by hydrogen sulfide aerosol. Furthermore, ovalbumin-induced transdifferentiation of pulmonary fibroblasts into myofibroblasts was reversed. Finally, hydrogen sulfide did abrogate in vitro the degranulation of the mast cell-like RBL-2H3 cell line. Similarly to the in vivo experiments the inhibitory effect was present only when the cells were activated by antigen exposure. In conclusion, inhaled hydrogen sulfide improves lung function and inhibits bronchial hyper-reactivity by modulating mast cells and in turn fibroblast activation

Abstract: This study investigated the contribution of hydrogen sulfide to biological oxygen demand (BOD5) and chemical oxygen demand (COD) in wastewater effluents, and documented the effect of storage times and conditions on the BOD5 and COD of pH-adjusted sodium sulfide solutions as well as graywater wetland effluent. Initial COD measurements of sulfide solutions were 84-89% of the theoretical oxygen demand (ThOD), 1.996 mg O2/mg S, whereas unseeded BOD5 measurements were 55-77%. For sulfide solutions, all storage conditions led to declines of >15% (COD, BOD5), and >31% (sulfide). For wetland effluent, storage without headspace was effective in reducing COD losses (3.7%), compared to storage with headspace (17%), and affected changes in turbidity, UVA-254 and pH. The results suggest that storage times and conditions should be controlled and reported when reporting BOD5 and COD of sulfide-rich samples. Wetland models representing sulfate reduction as a method of COD removal may need to be reconsidered

Abstract: Autotrophic denitrification with sulphide using nitrate (R1) and nitrite (R2) as electron acceptor was investigated at bench scale. Different solids retention times (SRT) (5 and 20 d) have been tested in R1 while R2 was operated at SRT = 13 d. The results indicated that the process allows complete sulphide removal to be achieved in all tested conditions. Tested sulphide loads were estimated from the H2S produced in a pilot-scale anaerobic digester treating vegetable tannery primary sludge; nitrogen loads originated from the nitrification of the supernatant. Average nitrogen removal efficiencies higher than 80% were observed in all the tested conditions once steady state was reached. A maximum specific nitrate removal rate equal to 0.35 g N-NO3(-) g VSS(-1) d(-1) was reached in R1. Due to sulphide limitation, incomplete denitrification was observed and nitrite and thiosulphate tend to accumulate especially in the presence of variable environmental conditions in both R1 and R2. Lower SRT caused higher NO2(accumulated)/NO3(reduced) ratios (0.22 and 0.24, with SRT of 5 d and 20 d, respectively) using nitrate as electron acceptor in steady-state condition. Temperature decrease caused sudden NO2(accumulated)/NO3(reduced) ratio increase in R1 and NO2(-) removal decrease in R2.

Abstract: Waste biomass from the industrial production of the amino acid L-cystine contains above-average concentrations of organic pollutants and significant concentrations of nitrogen and sulfur. The specific biogas production (SBP) of waste biomass was monitored in parallel suspended-growth laboratory anaerobic bioreactors. After severe inhibition was observed, three different procedures were applied to inhibited reactor sludge to counter-attack the inhibitory effects of sulfides, respectively hydrogen sulfide: micro-aeration, dilution with water and precipitation by ferrous iron cations. The performance of bioreactors was weekly monitored. Organic loading rates (as chemical oxygen demand, COD) ranged from 1.07 to 1.97 g L(-1) d(-1). At the end of the experimentation, SBP averaged 217, 300 and 320 l kg(-1) COD with a methane content of 21%, 52% and 54%; specific sludge production averaged 133, 111 and 400 g total solids kg(-1) COD, and inhibition was 49%, 27% and 25%; for the applied procedures of micro-aeration, dilution and precipitation respectively

Abstract: The gasotransmitter hydrogen sulfide (H2S) is emerging as a mediator of lung physiology and disease. Recent studies revealed that H2S administration limited perturbations to lung structure in experimental animal models of bronchopulmonary dysplasia (BPD), partially restoring alveolarization, limiting pulmonary hypertension, limiting inflammation, and promoting epithelial repair. No studies have addressed roles for endogenous H2S in lung development. H2S is endogenously generated by cystathionine beta-synthase (Cbs) and cystathionine gamma-lyase (Cth). We demonstrate here that the expression of Cbs and Cth in mouse lungs is dynamically regulated during lung alveolarization, and that alveolarization is blunted in Cbs/- and Cth/- mouse pups, where a 50% reduction in the total number of alveoli was observed, without any impact on septal thickness. Laser-capture microdissection and immunofluorescence staining indicated that Cbs and Cth were expressed in the airway epithelium and lung vessels. Loss of Cbs and Cth led to a 100-500% increase in the muscularization of small- and medium-sized lung vessels, which was accompanied by increased vessel wall thickness, and an apparent decrease in lung vascular supply. Ablation of Cbs expression using small interfering RNA, or pharmacological inhibition of Cth using propargylglycine in lung endothelial cells limited angiogenic capacity, causing a 30-40% decrease in tube length, and a 50% decrease in number of tubes formed. In
contrast, exogenous administration of H2S with GYY4137 promoted endothelial tube formation. These data confirm a key role for the H2S generating enzymes Cbs and Cth in pulmonary vascular development and homeostasis, and lung alveolarization.


Abstract: ETHNOPHARMACOLOGICAL RELEVANCE: Shunaoxin pill (SNX), one of the famous classical recipes in traditional Chinese medicine, is developed from the "Decoction of Xionggui". It has been used for treatment of cerebrovascular related diseases. It is well known that vasodilatation plays a very important role in cerebrovascular diseases. The effect of SNX on vasorelaxant activity has not yet been explored. Therefore, we aimed to investigate the vasorelaxant effects of SNX on isolated rat thoracic aorta so as to assess some of the possible mechanisms. We also investigate the gasotransmitter signaling pathway involved which has been rarely reported in isolated rat thoracic aorta before. AIM OF THE STUDY: The present study was performed to examine the vasodilative activity of SNX and its mechanisms in isolated rat thoracic aorta.

MATERIALS AND METHODS: SNX was studied on isolated rat thoracic aorta in vitro, including endothelium-intact and endothelium-denuded aortic rings. In present study, specific inhibitors including soluble guanylate cyclase (sGC) inhibitor 1 H-[1,2,4]oxadiazolo[4,3-a]quinoxaline-1-one (ODQ), cyclooxygenase (COX) inhibitor indomethacin (INDO), NO synthase inhibitor NG-nitro-l-arginine methyl ester (l-NAME), heme oxygenase-1 (HO-1) inhibitor zinc-protoporphyrin (ZnPP), cystathionine gamma-lyase (CSE) inhibitor DL-Propargylglycine (PAG), non-selective K+ channel inhibitor tetraethylammonium chloride (TEA), KV channel inhibitor 4-Aminopyridine (4-AP), and KATP channel inhibitor Glibenclamide (Gli) were used, they were added 20minutes before NE contraction and then added SNX to induce vasodilation. RESULTS: Removal of endothelium or pretreatment of aortic rings (intact endothelium) with l-NAME, ODQ or ZnPP significantly blocked SNX-induced relaxation. Pretreatment with the non-selective K+ channel inhibitor TEA, KV channel inhibitor 4-AP or the KATP channel inhibitor Gli, none of them had influences on the SNX-induced response (p>0.05). Besides, SNX inhibited the contraction triggered by NE in endothelium-denuded rings in Ca2+-free medium. SNX also produced rightward parallel displacement of CaCl2 curves.

CONCLUSIONS: These results suggest that SNX can induce less endothelium-dependent and more endothelium-independent vascular relaxation. The NO/cGMP and HO/CO pathways, blockade of Ca2+ channels are inhibition of IP3R mediated Ca2+ mobilization from intracellular stores, are likely involved in this relaxation. Furthermore the underlying mechanisms of combined compositions in SNX await further investigations.


Abstract: In addition to nitric oxide (NO), hydrogen sulfide (H2S) is recognized as a crucial gaseous messenger that exerts many biological actions in various tissues. An attempt was made to assess the roles and underlying mechanisms of both gases in isolated rat parotid acinar cells. Ductal cells and some acinar cells were found to express NO and H2S synthases. Cevimeline, a muscarinic receptor agonist upregulated endothelial NO synthase (eNOS) in parotid tissue. NO and H2S donors increased the intracellular Ca2+ concentration ([Ca2+]i). This was not affected by inhibitors of phospholipase C and inositol 1,4,5-trisphosphate receptors (InsP3Rs) but was decreased by blockers of RyRs, soluble guanylyl cyclase (sGC) and protein kinase G (PKG). The H2S donor evoked NO production, which was decreased by blockade of NO synthases or phosphoinositide-3-kinase (PI3K) or by hypotaurine, an H2S scavenger. The H2S donor-induced [Ca2+]i increase was diminished by an NO scavenger or the NO synthases blocker. These results suggest that NO and H2S play important roles in
regulating \([\text{Ca}^{2+}]_i\) via sGC-cGMP-PKG-RyRs but not via InsP3Rs. The effect of H2S may be partially through NO produced via PI3K-Akt-eNOS. It was concluded that both gases regulate \([\text{Ca}^{2+}]_i\) in a synergistic way mainly via RyRs in rat parotid acinar cells.

(55) Yuan B, Tang WH, Lu LJ, Zhou Y, Zhu HY, Zhou YL, et al. TLR4 upregulates CBS expression through NF-kappaB activation in a rat model of irritable bowel syndrome with chronic visceral hypersensitivity. World J Gastroenterol 2015 Jul 28;21(28):8615-28. Abstract: AIM: To investigate the roles of toll-like receptor 4 (TLR4) and nuclear factor (NF)-kappaB on cystathionine beta synthetase (CBS) expression and visceral hypersensitivity in rats. METHODS: This study used 1-7-wk-old male Sprague-Dawley rats. Western blot analysis was employed to measure the expression of TLR4, NF-kappaB and the endogenous hydrogen sulfide-producing enzyme CBS in colon dorsal root ganglia (DRG) from control and "irritable bowel syndrome" rats induced by neonatal colonic inflammation (NCI). Colon-specific DRG neurons were labeled with Dil and acutely dissociated to measure excitability with patch-clamp techniques. Immunofluorescence was employed to determine the co-expression of TLR4, NF-kappaB and CBS in Dil-labeled DRG neurons. RESULTS: NCI significantly upregulated the expression of TLR4 in colon-related DRGs (0.34 +/- 0.12 vs 0.72 +/- 0.02 for the control and NCI groups, respectively, P < 0.05). Intrathecal administration of the TLR4-selective inhibitor CLI-095 significantly enhanced the colorectal distention threshold of NCI rats. CLI-095 treatment also markedly reversed the hyperexcitability of colon-specific DRG neurons and reduced the expression of CBS (1.7 +/- 0.1 vs 1.1 +/- 0.04, P < 0.05) and of the NF-kappaB subunit p65 (0.8 +/- 0.1 vs 0.5 +/- 0.1, P < 0.05). Furthermore, the NF-kappaB-selective inhibitor pyrrolidine dithiocarbamate (PDTC) significantly reduced the upregulation of CBS (1.0 +/- 0.1 vs 0.6 +/- 0.1, P < 0.05) and attenuated visceral hypersensitivity in the NCI rats. In vitro, incubation of cultured DRG neurons with the TLR4 agonist lipopolysaccharide significantly enhanced the expression of p65 (control vs 8 h: 0.9 +/- 0.1 vs 1.3 +/- 0.1; control vs 12 h: 0.9 +/- 0.1 vs 1.3 +/- 0.1, P < 0.05; control vs 24 h: 0.9 +/- 0.1 vs 1.6 +/- 0.1, P < 0.01) and CBS (control vs 12 h: 1.0 +/- 0.1 vs 2.2 +/- 0.4; control vs 24 h: 1.0 +/- 0.1 vs 2.6 +/- 0.1, P < 0.05), whereas the inhibition of p65 via pre-incubation with PDTC significantly reversed the upregulation of CBS expression (1.2 +/- 0.1 vs 0.6 +/- 0.0, P < 0.01). CONCLUSION: Our results suggest that the activation of TLR4 by NCI upregulates CBS expression, which is mediated by the NF-kappaB signaling pathway, thus contributing to visceral hypersensitivity.

(56) Fonseca MD, Cunha FQ, Kashfi K, Cunha TM. NOSH-aspirin (NBS-1120), a dual nitric oxide and hydrogen sulfide-releasing hybrid, reduces inflammatory pain. Pharmacol Res Perspect 2015 Jun;3(3):e00133. Abstract: The development of nitric oxide (NO)- and hydrogen sulfide (H2S)-releasing nonsteroidal anti-inflammatory drugs (NSAIDs) has generated more potent anti-inflammatory drugs with increased safety profiles. A new hybrid molecule incorporating both NO and H2S donors into aspirin (NOSH-aspirin) was recently developed. In the present study, the antinociceptive activity of this novel molecule was compared with aspirin in different models of inflammatory pain. It was found that NOSH-aspirin inhibits acetic acid-induced writhing response and carrageenan (Cg)-induced inflammatory hyperalgesia in a dose-dependent (5-150 mumol/kg, v.o.) manner, which was superior to the effect of the same doses of aspirin. NOSH-aspirin's antinociceptive effect was also greater and longer compared to aspirin upon complete Freund's adjuvant (CFA)-induced inflammatory hyperalgesia. Mechanistically, NOSH-aspirin, but not aspirin, was able to reduce the production/release of interleukin-1 beta (IL-1beta) during Cg-induced paw inflammation. Furthermore, NOSH-aspirin, but not aspirin, reduced prostaglandin E2-induced hyperalgesia, which was prevented by treatment with a ATP-sensitive potassium channel (KATP) blocker (glibenclamide; glib.). Noteworthy, the antinociceptive effect of NOSH-aspirin was not associated with motor impairment. The present results indicate that NOSH-aspirin seems to present greater potency than aspirin to reduce inflammatory pain in several models. The enhanced
effects of NOSH-aspirin seems to be due to its ability to reduce the production of pronociceptive cytokines such as IL-1 beta and directly block hyperalgesia caused by a directly acting hyperalgesic mediator in a mechanism dependent on modulation of KATP channels. In conclusion, we would like to suggest that NOSH-aspirin represents a prototype of a new class of analgesic drugs with more potent effects than the traditional NSAID, aspirin


Abstract: OBJECTIVE: To examine the effect of H2S donor, sodium hydrosulfide (NaHS), on ET-1 level in plasma and aorta in rats with atherosclerosis (AS). METHOD: Thirty male rats, weighting 200-220 g, were randomly divided into AS, AS+NaHS and control groups, n = 10 in each group. Rats were given a single dose of vitamin D3 (700 000 U/kg) in the first three days and fed with a high-cholesterol diet for 8 weeks to induce AS. Rats in AS+NaHS group were intraperitoneally injected with an H2S donor NaHS, at a dose of 56 micromol/(kg.d) for 8 weeks. At the end of the experiment for 8 weeks, all the rats were sacrificed. The plasma was collected and the aorta and coronary tissues were isolated. The atherosclerotic lesions in both aorta and coronary arteries were detected using oil red O method. H2S concentration in plasma was determined with sulfide-sensitive electrode method. ET-1 levels in plasma and aorta were calculated by radioimmunoassay kit and the localization of ET-1 in the aorta was detected by immunohistochemistry. Plasma nitric oxide synthase (NOS), endothelial NOS (eNOS), inducible NOS (iNOS) were detected with colorimetry. RESULT: AS plaque area in root of aorta of rats in AS group, AS+NaHS group and control group were (11.6 +/- 3.3)%, (1.6 +/- 1.1)%, (0.0 +/- 0.1)% respectively. The difference in AS plaque area in root of aorta among the three groups was statistically significant (F=97.675, P < 0.05). AS plaque area in coronary artery of rats in AS group, AS+NaHS group and control group were (21.4 +/- 5.7)%, (4.8 +/- 2.5)% (0.0 +/- 0.0)% respectively. The difference in AS plaque area in coronary artery among the three groups was statistically significant (F=97.519, P < 0.05). Plasma H2S level in rats of AS group ((22.0 +/- 3.1) micromol/L) was significantly lower than that of control group ((27.9 +/- 1.0) micromol/L) and AS+NaHS group ((33.3 +/- 6.2) micromol/L, all P < 0.05). Compared with control group ((70.0 +/- 10.7) ng/L), plasma ET-1 in rats of AS group ((89.6 +/- 14.2) ng/L) and AS+NaHS group ((93.1 +/- 15.5) ng/L, P both < 0.05) were increased. However, there was no significant difference in plasma ET-1 content in rats between AS+NaHS group and AS group (P > 0.05). Compared with control group ((3.8 +/- 1.2) ng/g), ET-1 content in aorta in rats of AS group ((11.9 +/- 4.9) ng/g) and AS+NaHS group ((8.2 +/- 2.5) ng/g, both P < 0.05) were increased, and ET-1 content in aorta in rats of AS+NaHS group was decreased compared with AS group (P < 0.05). Immunohistochemistry results showed that ET expression in cytoplasm in aortic endothelial cells in rats of AS group was strengthened, while ET expression in rats of control group and AS+NaHS group was weak. NOS activity of rats in control group, AS group and AS+NaHS group was (25.4 +/- 5.6), (51.8 +/- 10.0) and (27.6 +/- 6.5) U/ml, eNOS activity (15.3 +/- 6.2), (4.5 +/- 2.7) and (8.7 +/- 3.9) U/ml, and iNOS activity (9.9 +/- 4.0), (47.3 +/- 10.7) and (19.0 +/- 5.2) U/ml, respectively. Differences among the three groups were statistically significant (NOS activity: F=37.231, P < 0.05, eNOS activity: F=14.600, P < 0.05, and iNOS activity: F=72.131, P < 0.05). CONCLUSION: H2S donor NaHS reduced the AS plaque in AS rats. The mechanisms might involve the protective effect of H2S on the vascular endothelial cell, decreasing ET-1 production in aortal endothelium of atherosclerotic rats


Abstract: Acidithiobacillus ferrooxidans strains were isolated from acidophilic microbial communities of Kazakhstan sulfide ore deposits. Their biotechnologically important
properties (optimal and maximal growth temperatures and resistance to NaCl) were determined. While temperature optima of the strains were the same (30-32 degrees C), temperature ranges were different. Thus, strain TFBK oxidized iron very poorly at 37 degrees C, while for strain TFV, the iron oxidation rate at this temperature was insignificantly lower than at lesser temperatures. NaCl inhibited the oxidative activity of both strains. Iron oxidation by strain TFV was inhibited at 5 g/L NaCl and was suppressed almost completely at 20 g/L. Iron oxidation by strain TFBK was inhibited by NaCl to a lesser degree, so that iron oxidation rate was relatively high at 10 g/L, while at 20 g/L NaCl the process was not suppressed completely, although the oxidation rate was low. Sulfur oxidation by these strains was less affected by NaCl than oxidation of ferrous iron. Sulfur oxidation by strain TFV was considerably inhibited only at 20 g/L NaCl, but was not suppressed completely. Sulfur oxidation by strain TFBK was more affected by NaCl. At 10 g/L NaCl the oxidation rate was much lower than at lower NaCl concentrations (sulfate concentrations after 6 days of oxidation at 5 and 10 g/L NaCl were -130 and -100 mM, respectively). While sulfur oxidation by strain TFBK was considerably inhibited at 10 and 20 g/L NaCl, similar to strain TFV it was not suppressed completely. Our results indicate the adaptation of the species A. ferrooxidans to a broad range of growth conditions.


Abstract: Quantum dots (QDots) are explored in biom edicine as highly fluorescent, photostable nanomaterials, but their use is impeded by their hydrophobic nature. In the present work, we evaluate the potential biomedical use of QDots that have been transferred into the aqueous phase by means of inorganic ligands. CdSe/CdS QDots were prepared and transferred to water upon ligand exchange to S(2-) ions. However, a multiparametric evaluation of the effect of these QDots on multiple cell types revealed significant QDot cytotoxicity. Using optimized methods, the QDots were found to rapidly degrade under endosomal pH, resulting in leached Cd(2+). Together with the induction of oxidative stress, this significantly affected cell viability. Using proliferation-restricted cells, QDot degradation was found to augment cytotoxicity with time resulting in mitochondrial and DNA damage, effects on cell morphology and cell functionality. The final non-cytotoxic concentration was defined at 2 nM, enabling cells to be tracked up to 2 cell divisions. A direct comparison with other QDots and fluorescent particles studied resulted in similar concentrations; however, the functionality of previously analyzed particles was much higher. These data reveal that comparing NP toxicity based on particle concentrations is extremely difficult. A comparison of NPs is better obtained by evaluating NP functionality using a straightforward approach, such as follow-up of QDot fluorescence in dividing cells. These data highlight the importance of (1) considering QDot stability in the intracellular microenvironment, (2) the protective nature of the QDot-stabilizing coating, (3) the need for comparison of particle functionality to understand any observed effects.


Abstract: Animal feeding operations (AFOs) emit various air pollutants, including ammonia, hydrogen sulfide, particulate matter, volatile organic compounds, methane, and nitrous oxide. Several of these pollutants are regulated under federal clean air statutes, yet AFOs have largely escaped regulation under these laws because of challenges in accurately estimating the rate and quantity of emissions from various types of livestock operations. Recent Environmental Protection Agency (EPA) efforts to collect emissions data, develop an emissions model capable of estimating emissions at AFOs nationwide, and establish emissions estimating methodologies for certain key livestock air pollutants suffered from design flaws and omitted pollutants of concern. Moreover, this process seems to have stalled, delaying other regulatory reforms needed to increase
transparency and increase regulation of these facilities. Until EPA establishes these methodologies, significant AFO pollution regulation under the Clean Air Act or emissions reporting statutes will be very difficult to achieve, and the public health and environmental impacts of these emissions will continue unabated.

Abstract: Biodiversity of sulfate-reducing bacterial communities in the water column of the Gdansk Deep, Baltic Sea, where H2S had been detected in near-bottom layers, was analyzed by PCR with primers for the 16S rRNA genes of six major phylogenetic subgroups of sulfate-reducing bacteria (SRB). Using denaturing gradient gel electrophoresis followed by sequencing, the nucleotide sequences of reamplified dsrB gene fragments from investigated water samples were determined. For the first time the presence of nucleotide sequences of the dsrB gene was detected by PCR in the water samples from all hydrochemical layers, including subsurface oxic waters. The presence of the 16S rRNA genes of representatives of Desulfothermus, Desulfococcus-Desulfonema-Desulfosarcina, and Desulfovibrio-Desulfomicrobium SRB subgroups was also revealed throughout the water column of the Gdansk Deep. Analysis of translated amino acid sequences encoded by the dsrB gene demonstrated the highest homology with the relevant sequences of uncultured SRB from various marine habitats.

Abstract: T-type Ca(2+) channels are a distinct family of low voltage-activated Ca(2+) channels which serve many roles in different tissues. Several studies have implicated them, for example, in the adaptive responses to chronic hypoxia in the cardiovascular and endocrine systems. Hydrogen sulfide (H2S) was more recently discovered as an important signalling molecule involved in many functions, including O2 sensing. Since ion channels are emerging as an important family of target proteins for modulation by H2S, and both T-type Ca(2+) channels and H2S are involved in cellular responses to hypoxia, we have investigated whether recombinant and native T-type Ca(2+) channels are a target for modulation by H2S. Using patch-clamp electrophysiology, we demonstrate that the H2S donor, NaHS, selectively inhibits Cav3.2 T-type Ca(2+) channels heterologously expressed in HEK293 cells, whilst Cav3.1 and Cav3.3 channels were unaffected. Sensitivity of Cav3.2 channels to H2S required the presence of the redox-sensitive extracellular residue H191, which is also required for tonic binding of Zn(2+) to this channel. Chelation of Zn(2+) using TPEN prevented channel inhibition by H2S. H2S also selectively inhibited native T-type channels (primarily Cav3.2) in sensory dorsal root ganglion neurons. Our data demonstrate a novel target for H2S regulation, the T-type Ca(2+) channel Cav3.2. Results have important implications for the proposed pro-nociceptive effects of this gasotransmitter. Implications for the control of cellular responses to hypoxia await further study.

Abstract: Although the gasotransmitter hydrogen sulfide (H2S) generally dilates systemic arteries in mammals, it causes constriction of pulmonary arteries. In isolated rat pulmonary arteries, we have shown that the H2S precursor cysteine enhances both hypoxic pulmonary vasoconstriction and tension development caused by the agonist prostaglandin F2alpha under normoxic conditions. These effects were blocked by propargylglycine (PAG), a blocker of the enzyme cystathionine gamma lyase which metabolises cysteine to sulfide. In the present study, we evaluated whether 3-mercaptoppyruvate (3-MP), a sulfide precursor which is thought to give rise to sulfide when it is metabolised by the enzyme mercaptoppyruvate sulfotransferase, also
enhanced contraction. Application of 3-MP prior to hypoxic challenge caused a marked enhancement of HPV which was completely blocked by both L- and D,L-PAG (both 1 mM). Cumulative application of 3-1,000 μM 3-MP during an ongoing contraction to PGF2α under normoxic conditions also caused a marked increase in tension. Application of D-cysteine (1 mM) also enhanced HPV, and this effect was prevented by both the D-amino acid oxidase inhibitor sodium benzoate (500 μM) and 1 mM L-PAG

Abstract: The cascade of transduction of hypoxia and hypercapnia, the natural stimuli to chemoreceptor cells, is incompletely understood. A particular gap in that knowledge is the role played by second messengers, or in a most ample term, of modulators. A recently described modulator of chemoreceptor cell responses is the gaseous transmitter hydrogen sulfide, which has been proposed as a specific activator of the hypoxic responses in the carotid body, both at the level of the chemoreceptor cell response or at the level of the global output of the organ. Since sulfide behaves in this regard as cAMP, we explored the possibility that sulfide effects were mediated by the more classical messenger. Data indicate that exogenous and endogenous sulfide inhibits adenyl cyclase finding additionally that inhibition of adenyl cyclase does not modify chemoreceptor cell responses elicited by sulfide. We have also observed that transient receptor potential cation channels A1 (TRPA1) are not regulated by sulfide in chemoreceptor cells

Abstract: The physiological and pathological roles of hydrogen sulfide (H2S) in the regulation of cardiovascular functions have been recognized. H2S protects against the hypoxia/reoxygenation (H/R)-induced injury and apoptosis of cardiomyocytes, and ischemic post-conditioning (PC) plays an important role in cardioprotection from H/R injury in neonatal cardiomyocytes but not in aging cardiomyocytes. Whether H2S is involved in the recovery of PC-induced cardioprotection in aging cardiomyocytes is unclear. In the present study, we found that both H/R and PC decreased cystathionine-gamma-lyase (CSE) expression and the production rate of H2S. Supplementation of NaHS protected against H/R-induced apoptosis, the expression of cleaved caspase-3 and cleaved caspase-9, the release of cytochrome c (Cyt c), and mPTP opening. The addition of NaHS also counteracted the reduction of cell viability caused by H/R and increased the phosphorylation of ERK1/2, PI3K, Akt, GSK-3beta and mitochondrial membrane potential. Additionally, NaHS increased Bcl-2 expression, promoted PKC-epsilon translocation to the cell membrane, and activated mitochondrial ATP-sensitive K channels (mitoKATP). PC alone did not provide cardioprotection in H/R-treated aging cardiomyocytes, which was significantly restored by the supplementation of NaHS. In conclusion, our results suggest that exogenous H2S restores PC-induced cardioprotection via the inhibition of mPTP opening by the activation of the ERK1/2-GSK-3beta, PI3K-Akt-GSK-3beta and PKC-epsilon-mitoKATP pathways in aging cardiomyocytes. These findings provide a novel target for the treatment of aging ischemic cardiomyopathy

Abstract: Hydrogen sulfide (H2S) has been shown to have a prominent role in the regulation of reproductive system function and fertility. The aim of the study was to assess the effect of a H2S donor, sodium hydrosulfide (NaHS), on mouse sperm migration in vitro. Special plates with 4 corner wells filled with balanced salt solution (control) and various NaHS solutions in concentrations of 2.5 mmol/l, 5 mmol/l or 10
mmol/l were applied. Spermatozoa from each male mouse were injected (the experiment was repeated with ten BALB/c 5-month old males) into the central pocket, connected with the wells with ducts. After 1 h, 2 h and 4 h of incubation, the number of spermatozoa in each well was determined using Burker's counting chambers. The number of spermatozoa in all corner wells were summed and the number of the cells in each well was treated as the percentage share of all the migrated spermatozoa. At the time points of 1 hour and 4 hours, no differences regarding chemotactic features of spermatozoa to the utilized solutions were observed. After two hours of incubation the attenuating effect of NaHS medium and high level solutions on spermatozoa migration was observed, but not for the low concentration mixture: H(3, N = 40) = 9.65, P = 0.022; control group vs 5 mmol/l NaHS solution: 36.0% vs 18.5%, P = 0.023; control group vs 10 mmol/l NaHS solution group: 36.0% vs 17.0%, P 0.011. In conclusion, NaHS has a transitional attenuating effect on spermatozoa migration in vitro.

Abstract: We used a novel amperometric microsensor for measuring hydrogen gas production and consumption at high spatio-temporal resolution in cyanobacterial biofilms and mats dominated by non-heterocystous filamentous cyanobacteria (Microcoleus chthonoplastes and Oscillatoria sp.). The new microsensor is based on the use of an organic electrolyte and a stable internal reference system and can be equipped with a chemical sulfide trap in the measuring tip; it exhibits very stable and sulfide-insensitive measuring signals and a high sensitivity (1.5-5 pA per mumol L(-1) H2). Hydrogen gas measurements were done in combination with microsensor measurements of scalar irradiance, O2, pH, and H2S and showed a pronounced H2 accumulation (of up to 8-10% H2 saturation) within the upper mm of cyanobacterial mats after onset of darkness and O2 depletion. The peak concentration of H2 increased with the irradiance level prior to darkening. After an initial build-up over the first 1-2 h in darkness, H2 was depleted over several hours due to efflux to the overlaying water, and due to biogeochemical processes in the uppermost oxic layers and the anoxic layers of the mats. Depletion could be prevented by addition of molybdate pointing to sulfate reduction as a major sink for H2. Immediately after onset of illumination, a short burst of presumably photo-produced H2 due to direct biophotolysis was observed in the illuminated but anoxic mat layers. As soon as O2 from photosynthesis started to accumulate, the H2 was consumed rapidly and production ceased. Our data give detailed insights into the microscale distribution and dynamics of H2 in cyanobacterial biofilms and mats, and further support that cyanobacterial H2 production can play a significant role in fueling anaerobic processes like e.g., sulfate reduction or anoxygenic photosynthesis in microbial mats.

Abstract: The aim of this study was to assess the effects of hydrogen sulfide on high glucose-induced mouse podocyte (MPC) injury and the underlying mechanisms. Mouse podocytes were randomly divided into 4 groups, including high glucose (HG), normal glucose (NG), normal glucose + DL-propargylglycine (PPG), and high glucose + NaHS (HG + NaHS) groups for treatment. Then, ZO-2, nephrin, beta-catenin, and cystathionine gamma-lyase (CSE) protein expression levels were determined by western blot. We found that high glucose significantly reduced nephrin, ZO-2, and CSE expression levels (P<0.05), and overtly elevated beta-catenin amounts (P<0.05), in a time-dependent manner. Likewise, PPG at different concentrations in normal glucose resulted in significantly lower CSE, ZO-2, and nephrin levels (P<0.05), and increased beta-catenin amounts (P<0.05). Interestingly, significantly increased ZO-2 and nephrin levels, and overtly reduced beta-catenin amounts were observed in the HG + NaHS group compared with HG treated cells (P<0.01). Compared with NG treated cells, decreased ZO-2 and
nephrin levels and higher beta-catenin amounts were obtained in the HG + NaHS group. In conclusion, CSE downregulation contributes to hyperglycemia induced podocyte injury, which is alleviated by exogenous H2S possibly through ZO-2 upregulation and the subsequent suppression of Wnt/beta-catenin pathway

(69) Xu Y, Dai X, Zhu D, Xu X, Gao C, Wu C. An exogenous hydrogen sulphide donor, NaHS, inhibits the apoptosis signaling pathway to exert cardio-protective effects in a rat hemorrhagic shock model. Int J Clin Exp Pathol 2015;8(6):6245-54. Abstract: Hydrogen sulfide (H2S) has been reported to be intertwined in multiple systems, specifically in the cardiovascular system. However, the mechanisms underlying remain controversial. In the present study, we assessed the cardio-protective effects of H2S in the rat hemorrhagic shock model. Hemorrhagic shock was induced in adult male Sprague-Dawley rats by drawing blood from the femoral artery to maintain the mean arterial pressure at 35-40 mmHg for 1.5 h. The rats were assigned to four groups and the H2S donor, NaHS (28 mumol/kg, i.p.), was injected before the resuscitation in certain groups. After resuscitation the animals were observed and then killed to harvest the hearts. The morphological investigation and ultrastructural analyses were done and apoptotic cells were detected. The levels of relevant proteins were examined using Western blotting and immunohistochemical analyses. Resuscitated hemorrhagic shock induced heart injury and significantly increased the levels of serum myocardial enzymes, creatine kinase (CK) and lactate dehydrogenase (LDH) levels. Furthermore, it caused marked increase of apoptotic cells in heart tissue. Moreover, the expression of death receptor Fas and Fas-ligand, as well as the expression of apoptosis-relevant proteins active-caspase 3 and active-caspase 8 were markedly increased. Administration of NaHS significantly ameliorated hemorrhagic shock caused hemodynamic deterioration, decreased myocardial enzymes elevation, protected myocardial ultrastructure, and inhibited the expression of apoptosis-relevant proteins. It suggested that H2S might exert its cardio-protective roles via both the extrinsic Fas/FasL/caspase-8/caspase-3 pathway and the intrinsic mitochondria-involved pathways

(70) Kwan S, Boudes E, Benseler A, Gilbert G, Saint-Martin C, Shevell M, et al. Evolution of Apparent Diffusion Coefficient and Fractional Anisotropy in the Cerebrum of Asphyxiated Newborns Treated with Hypothermia over the First Month of Life. Neural Plast 2015;2015:653727. Abstract: The objective of this study was to assess the evolution of diffusion-weighted imaging (DWI) and diffusion-tensor imaging (DTI) over the first month of life in asphyxiated newborns treated with hypothermia and to compare it with that of healthy newborns. Asphyxiated newborns treated with hypothermia were enrolled prospectively; and the presence and extent of brain injury were scored on each MRI. Apparent diffusion coefficient (ADC) and fractional anisotropy (FA) values were measured in the basal ganglia, in the white matter and in the cortical grey matter. Sixty-one asphyxiated newborns treated with hypothermia had a total of 126 ADC and FA maps. Asphyxiated newborns developing brain injury eventually had significantly decreased ADC values on days 2-3 of life and decreased FA values around day 10 and 1 month of life compared with those not developing brain injury. Despite hypothermia treatment, asphyxiated newborns may develop brain injury that still can be detected with advanced neuroimaging techniques such as DWI and DTI as early as days 2-3 of life. A study of ADC and FA values over time may aid in the understanding of how brain injury develops in these newborns despite hypothermia treatment

of low body temperature called ‘torpor’ without signs of organ injury. Recently, we identified an essential role of hydrogen sulfide (H2S) in entrance into torpor and preservation of kidney integrity during hibernation. A torpor-like state can be induced pharmacologically by injecting 5'-Adenosine monophosphate (5'-AMP). The mechanism by which 5'-AMP leads to the induction of a torpor-like state, and the role of H2S herein, remains to be unraveled. Therefore, we investigated whether induction of a torpor-like state by 5-AMP depends on H2S production. METHODS: To study the role of H2S on the induction of torpor, amino-oxyacetic acid (AOAA), a non-specific inhibitor of H2S, was administered before injection with 5'-AMP to block endogenous H2S production in Syrian hamster. To assess the role of H2S on maintenance of torpor induced by 5'-AMP, additional animals were injected with AOAA during torpor. KEY RESULTS: During the torpor-like state induced by 5'-AMP, the expression of H2S-synthesizing enzymes in the kidneys and plasma levels of H2S were increased. Blockade of these enzymes inhibited the rise in the plasma level of H2S, but neither precluded torpor nor induced arousal. Remarkably, blockade of endogenous H2S production was associated with increased renal injury. CONCLUSIONS: Induction of a torpor-like state by 5'-AMP does not depend on H2S, although production of H2S seems to attenuate renal injury. Unraveling the mechanisms by which 5'-AMP reduces the metabolism without organ injury may allow optimization of current strategies to limit (hypothermic) IRI and improve outcome following organ transplantation, major cardiac and brain surgery.

Abstract: To identify novel susceptibility variants for osteoporosis in Korean postmenopausal women, we performed a genome-wide association analysis of 1180 nonsynonymous single nucleotide polymorphisms (nsSNPs) in 405 individuals with osteoporosis and 722 normal controls of the Korean Association Resource cohort. A logistic regression analysis revealed 72 nsSNPs that showed a significant association with osteoporosis (p<0.05). The top 10 nsSNPs showing the lowest p-values (p = 5.2x10^-4-8.5x10^-3) were further studied to investigate their effects at the protein level. Based on the results of an in silico prediction of the protein's functional effect based on amino acid alterations and a sequence conservation evaluation of the amino acid residues at the positions of the nsSNPs among orthologues, we selected one nsSNP in the SQRDL gene (rs1044032, SQRDL I264T) as a meaningful genetic variant associated with postmenopausal osteoporosis. To assess whether the SQRDL I264T variant played a functional role in the pathogenesis of osteoporosis, we examined the in vitro effect of the nsSNP on bone remodeling. Overexpression of the SQRDL I264T variant in the preosteoblast MC3T3-E1 cells significantly increased alkaline phosphatase activity, mineralization, and the mRNA expression of osteoblastogenesis markers, Runx2, Sp7, and Bglap genes, whereas the SQRDL wild type had no effect or a negative effect on osteoblast differentiation. Overexpression of the SQRDL I264T variant did not affect osteoclast differentiation of the primary-cultured monocytes. The known effects of hydrogen sulfide (H2S) on bone remodeling may explain the findings of the current study, which demonstrated the functional role of the H2S-catalyzing enzyme SQRDL I264T variant in osteoblast differentiation. In conclusion, the results of the statistical and experimental analyses indicate that the SQRDL I264T nsSNP may be a significant susceptibility variant for osteoporosis in Korean postmenopausal women that is involved in osteoblast differentiation.

Abstract: The data of foreign studies over the last 15 years devoted to endogenous synthesis and biological role of hydrogen sulfide in micromolar quantities which complemented the already two well-known gas transmitters - OH and NO are presented in this review. Despite the short period since the physiological properties of hydrogen...
sulfide were opened (about 20 years) it was found that this gas transmitter plays a key role in the regulation of nerve (neural signal transmission), cardiovascular (relaxation of smooth muscles), immune (anti-inflammatory and cytoprotective agent) sensory, gastrointestinal (output of insulin) systems and in the metabolism of various organs. Currently the role of H2S in the pathogenesis of different diseases, neurodegenerative diseases, diabetes, heart failure) is being studying. The developments of drugs that act as either exogenous donors H2S or blockers of the biosynthesis of H2S are promising. With consideration the fact that H2S is a representative of non-synaptic way of intercellular communication based on diffusion of molecules of inorganic compounds in the intercellular space in all directions and effect on distant from their place of formation non-synaptic receptors it is suggested to use exogenous H2S in strict proportion for the treatment of a number of human diseases