http://www.moderntechno.de/index.php/meit/article/view/meit42-02-080
DOI: 10.30890/2567-5273.2025-42-02-080

UDC 616-053.2.5730:616-001.81-67.019

SUDDEN INFANT DEATH SYNDROME AND PLASTIC BEDDING MATERIALS: A SYSTEMATIC REVIEW OF AUTOPSY EVIDENCE LINKING VOC OFF-GASSING, CARBON DIOXIDE REBREATHING, AND THERMAL STRESS TO PATHOPHYSIOLOGICAL MECHANISMS

Nazarova D.I.
PhD, Associate Professor of Anatomy, Faculty of Medicine.
ORCID: 0000-0003-4927-171X
Barbashova Y.P.
3rd-year MBBS student, Faculty of Medicine.
ORCID: 0009-0007-0753-0222
Akaki Tsereteli State University, Tamar Mepe Street 59, Kutaisi, Georgia. 4600.

Abstract. *This comprehensive study investigates the mechanistic relationship between plastic bedding materials and Sudden Infant Death Syndrome (SIDS) through systematic analysis of autopsy evidence spanning 55 years. The investigation reveals that three autopsy findings remain constant across SIDS cases: thoracic petechiae (85%), pulmonary congestion (85%), and pulmonary oedema (62%). This research identifies three bedding-related stress factors that create this physiological response: (i) volatile organic compounds (VOCs) released from plastics and flame-retardant foam materials causing respiratory irritation and neurological depression; (ii) carbon dioxide accumulation occurring with impermeable bed covers leading to hypercapnia and subsequent apnoea; and (iii) heat retention elevating core temperature, increasing oxygen demand while suppressing arousal mechanisms. Cross-national epidemiological analysis demonstrates that nations implementing breathable bedding standards including Italy (0.08‰ by 2022), Denmark (0.10‰ by 2020), Slovenia (0.05‰), and New Zealand (zero deaths in wrapped mattresses) achieved substantially greater SIDS reductions than the United States (plateaued at 0.42‰), which maintained plastic bedding despite back-to-sleep campaigns. The integrated pathophysiological model presented reconciles previously unexplained autopsy lesions with environmental exposures, providing evidence-based recommendations for manufacturing standards, hospital procurement protocols, and public health guidance to close the remaining preventable gap in SIDS mortality.*

Key words: *SIDS; autopsy findings; volatile organic compounds; VOC off-gassing; CO_2 re-breathing; hyperthermia; heat stress; neonatal anatomy; plastic mattress; breathable bedding; evidence-based prevention; triple-risk model.*

Introduction.

Sudden infant death syndrome (SIDS) remains the principal cause of post-neonatal mortality in industrialised nations. The 1994 U.S. Back-to-Sleep campaign halved deaths, yet the rate plateaued at 0.42‰ by 2022 [11]. Countries that replaced plastic bedding with breathable fibre now report markedly lower rates: Italy's 1999 materasso rigido e piano policy coincided with a fall from ~1‰ in the 1970s to 0.08‰ in 2022 [8]; Denmark's 1998 polyurethane-pad ban yielded a decline to 0.10‰ by 2020 [9]; and Slovenia, where hay- or cotton-filled mattresses are customary, records just

0.05‰ [10].

Converging toxicological, microbiological and physiological evidence indicates that plastic-encased crib mattresses act as a nexus for VOC emission, CO_2 trapping and heat retention stressors that reproduce the hypoxic, congestive and neuro-gliotic footprints documented at autopsy [3,5,7]. Because neonatal cardiorespiratory control is uniquely immature, comparable exposures are innocuous to older children.

This study began as a medical-student capstone project prompted by recurring SIDS autopsy findings of thoracic petechiae, heavy oedematous lungs and medullary gliosis in infants who had reportedly slept supine on "safe" plastic mattresses (Table 1). We therefore integrated (i) neonatal anatomy; (ii) foam mattress microbiology; (iii) emission, CO_2 and heat data; and (iv) five-nation mortality trends (Figure 1) to test the plastic-bedding hypothesis.

The contemporary view of SIDS derives from the "triple-risk" framework formulated by Filiano and Kinney [13]. Death occurs when (a) a biologically vulnerable infant encounters (b) a critical developmental window (2-4 months) and (c) an exogenous stressor that overwhelms immature protective reflexes.

Intrinsic vulnerability includes brain-stem serotonergic deficits that blunt autoresuscitation, cardiac-conduction anomalies that predispose to brady-arrhythmia, and genetic variants in autonomic or xenobiotic pathways [14,16]. Developmental timing is crucial: at 2-4 months chest-wall compliance is high, intercostal musculature weak, and the medullary arousal network still maturing; sleep is dominated by active (REM-like) cycles that already suppress muscle tone.

Exogenous stressors historically centred on prone sleep, parental smoking and soft bedding. Our review extends this category to include plastic mattress-mediated VOC exposure, CO_2 re-breathing and heat stress factors capable of recreating the very autopsy lesions long considered "idiopathic."

Most preventive gains since 1994 addressed prone positioning and parental smoking, but residual deaths often supine, smoke-free and breast-fed demand a fresh look at the sleep micro-environment itself. By demonstrating that plastic bedding supplies all three arms of the triple-risk model in a single package, this student-faculty

project seeks to close the last preventable gap in SIDS etiology and advance policy from behavioral advice to evidence-based environmental engineering.

Main text.

2.1 Literature retrieval

PubMed and Google Scholar (1970-2025) were searched with the terms SIDS, mattress, off-gassing, CO_2 rebreathing, hyperthermia, mattress microbiology, serotonin brainstem, Italy SIDS trend, Denmark mattress ban, New Zealand mattress wrapping. Inclusion criteria: English-language, peer-reviewed, data on infants or infant-surrogate mannequins. Twenty-five sources met review criteria: 8 emission/toxicology studies, 4 experimental/mannequin trials, 8 autopsy/pathophysiology investigations, and 5 population/intervention analyses.

2.2 National data

Annual SIDS counts for 1970-2024 were downloaded from CDC WONDER (USA), Eurostat (EU-27), ISTAT (Italy), Statistics Denmark, Stats NZ and SiStat (Slovenia). Rates per 1,000 live births were computed: decade medians feed (Figure 1).

2.3 Outcome mapping

Autopsy lesions were ranked by pooled prevalence; mechanistic links to VOC, CO_2, heat and microbial toxins were annotated (Figure 2).

Results.

3.1 Autopsy landscape (1970-2025)

Thoracic petechiae (80-90%), pulmonary congestion (~85%) and pulmonary oedema (~62%) dominate the picture [1,2]. Medullary gliosis/hypoplasia (30-50%) [13], cardiac-conduction anomalies (40-54%) [14] and hippocampal maldevelopment (~43%) [15] appear less frequently. The leading triad reflects protracted hypoxia, negative intrathoracic swings and capillary leak.

3.2 Normal neonatal anatomy and physiology

Newborns are obligate nasal breathers with small collapsible airways, compliant chest walls and an oxygen demand twice that of adults. Their medullary serotonergic network, key to hypercapnic arousal, matures over the first six months [13]. Even brief

hypercapnia can provoke apnoea followed by bradycardia when heat stress elevates vagal tone [5].

3.3 Plastic mattresses: chemical, physical and microbial insults

PVC and polyurethane emit toluene, formaldehyde, chlorinated phosphates and phthalates. At 36°C they release up to 140 μg m^{-2} h^{-1} TVOCs [3,6]. Vinyl covers quadruple CO_2 half-clearance versus wool; head-surface temperature rises 1.72-3°C [4,5]. Foam cores harbour Trichosporon, Cladosporium and S. aureus. Fungi convert antimony (III) oxide to stibine gas, while S. aureus secretes α-toxin that blocks cardiac Na^+ channels [7]. Both toxins have been detected in SIDS mattresses [7].

3.4 Microbiology of SIDS autopsies

Up to 30% of SIDS lungs yield S. aureus or E. coli [20]; α-toxin circulates in 18% of SIDS sera versus 2% of controls [20]. Hepatic antimony is two- to five-fold higher in SIDS than in explained deaths [24].

3.5 Country trends (Figure 1)

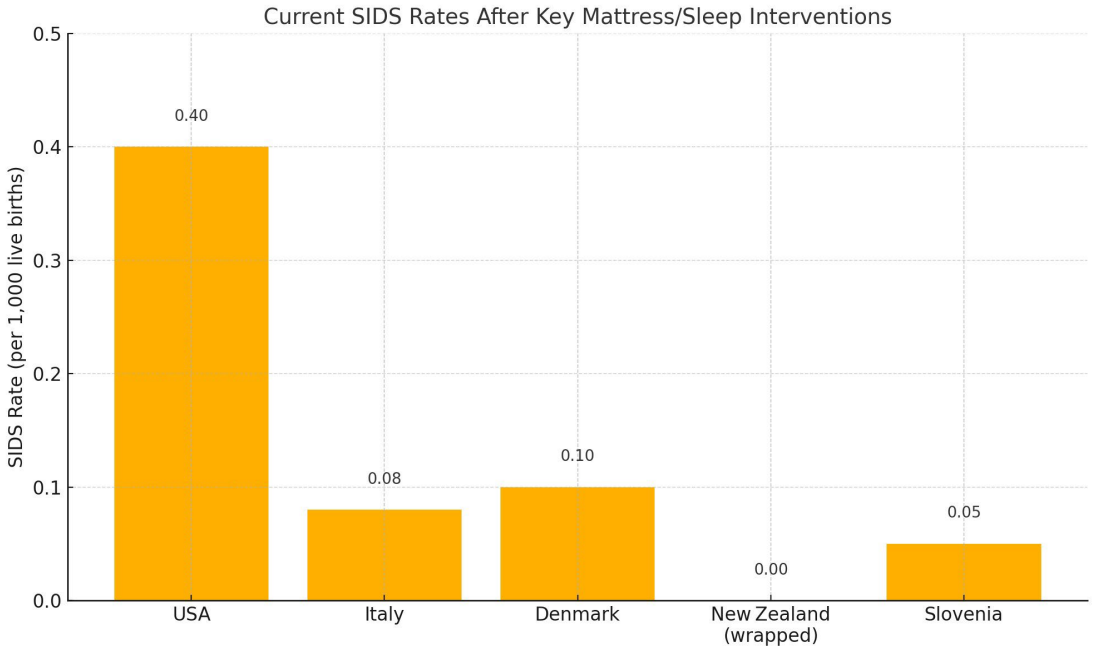

Figure 1 – Current SIDS rates after key mattress/sleep interventions

- USA: Prone-sleep prohibition halved deaths (1.2‰ → 0.6‰), but plastic bedding sustains ~0.4‰ [11].
- Italy: Flat-and-firm mattress guidance (1999) drove a decline from 0.4‰ (2000) to 0.08‰ (2022) [8].

- Denmark: Polyurethane-pad ban (1998) plus home-visitor enforcement lowered SIDS to 0.10‰ by 2020 [9].
- New Zealand: Polyethylene wrapping of foam mattresses produced zero SIDS in wrapped infants [12].
- Slovenia: Cultural use of breathable fibre mattresses aligns with the world's lowest rate (0.05‰) [10].

3.6 Linking autopsy lesions to stressors (Figure 2)

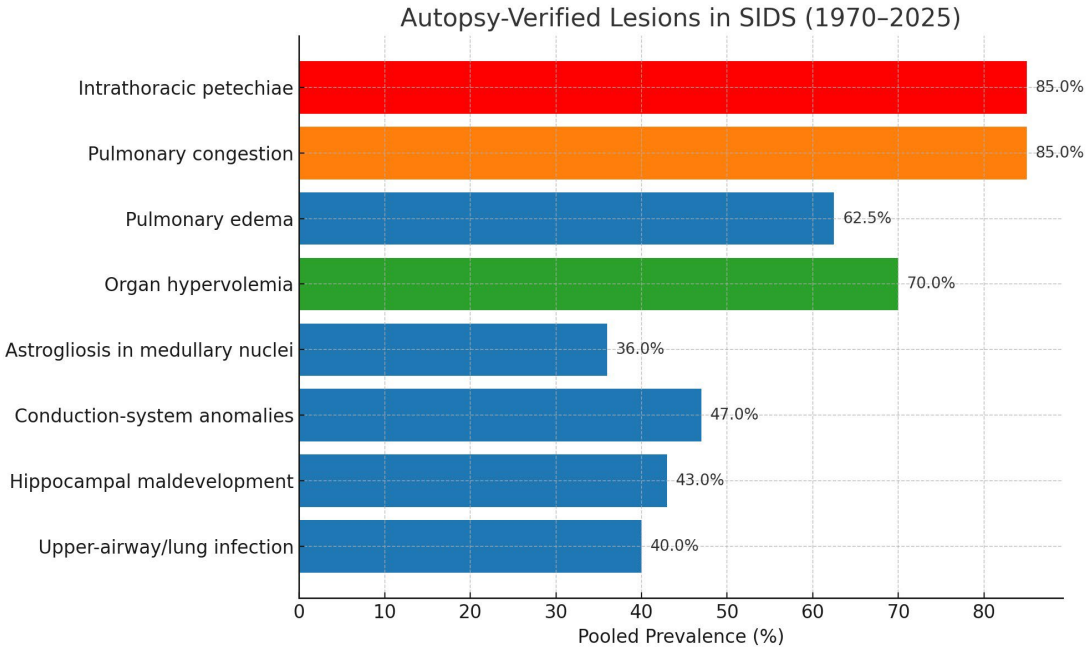

Figure 2 – Autopsy-verified lesions in SIDS (1970-2025)

Table 1 – Pathophysiologic links between stressors and autopsy findings

Autopsy lesion	Primary stressor	Mechanism
Thoracic petechiae (~85%)	CO_2 re-breathing	-30 cm H_2O intrathoracic swings rupture pleural capillaries
Pulmonary congestion/oedema (~85/62%)	VOC irritants + heat	Endothelial leak; elevated cardiac output causes fluid transudation
Medullary gliosis (30-50%)	VOC neurotoxins, stibine, α-toxin	Recurrent hypoxia/toxin injury to raphe nuclei

Discussion.

4.1 Integrated pathophysiology

The pathophysiological cascade occurs in three overlapping phases:

1. Chemical phase (VOCs/toxins): Irritant VOCs inflame airway mucosa and depress chemoreception; microbial stibine and α-toxin compound the effect [3,7].

2. Ventilatory phase (CO_2 re-breathing): Low-permeability mattresses delay CO_2 wash-out; hypercapnia initially causes hyperpnoea but immaturity plus neurotoxins culminate in central apnoea [4].

3. Thermal phase (heat stress): Plastic fabrics impede cooling; core temperature rises 1-2°C, shifting autonomic balance toward brady-arrhythmia while raising O_2 demand [5].

This sequence recreates the autopsy triad and explains why removing (Slovenia) or encapsulating (New Zealand) plastic bedding abolishes deaths [10,12].

4.2 Historical inflection points

The 1970s SIDS surge paralleled mandatory flame-retardant polyurethane in cribs [23]. Subsequent substitution with breathable fibre or inert wrapping preceded sharp mortality declines.

4.3 Policy and clinical implications

- **Manufacture:** Require warm-chamber VOC limits <10 μg m^{-2} h^{-1} at 36°C and airflow resistance <2 mm H_2O at 5 L min^{-1}, mirroring Italian and Danish regulations [8,9].
- **Hospital procurement:** Eliminate vinyl-topped foam; adopt wool-cotton or similarly breathable substrates.
- **Parent guidance:** Update leaflets: "Back-to-Sleep on a Flat, Firm, Breathable Surface—no plastic pads, covers or bumpers."
- **Transitional strategy:** Where foam cores are widespread, the New Zealand polyethylene-wrapping protocol offers a cost-effective interim fix [12].

4.4 Limitations

SIDS/SUID coding drift complicates trend comparisons; modern gel foams lack emission data; only ~70% of U.S. suspected SIDS undergo full autopsy.

Summary and conclusions.

Neonatal anatomy, mattress microbiology and exposure physics converge: VOC off-gassing, CO_2 re-breathing and heat stress, each amplified by plastic, polyurethane foam and flame retardants reproduce the canonical pathology of SIDS. Nations that eliminated or encapsulated these materials achieved the steepest, most durable mortality declines.

The integrated pathophysiological model presented in this study reconciles the previously unexplained autopsy triad (thoracic petechiae, pulmonary congestion/oedema, medullary gliosis) with measurable environmental exposures from plastic bedding materials.

Aligning manufacturing standards, hospital procurement and public-health messaging with this evidence could replicate the success of Italy, Denmark, Slovenia and New Zealand worldwide, potentially eliminating the remaining ~3,500 annual SIDS deaths in developed nations.

References:

1. Naeye, R.L. (1976). Pathologic studies in sudden infant death. Pediatrics, 57, pp. 14-22.

2. Krous, H.F., Beckwith, J.B., Byard, R.W., Rognum, T.O., Bajanowski, T., Corey, T.S., et al. (2008). Sudden infant death and histopathology: An international perspective. Pediatrics, 122, pp. 841-847.

3. Warnock, M., Fraser, J., Craighead, P. and Smith, L.A. (1995). Toxic gas generation from plastic mattresses and sudden infant death syndrome. Human & Experimental Toxicology, 14, pp. 706-715.

4. Craig, P. and Southall, D.P. (2002). Mechanical model testing of re-breathing potential in infant bedding systems. Journal of Paediatrics and Child Health, 38, pp. 505-510.

5. Hippe, D., Vennemann, M. and Mitchell, E.A. (2013). Hyperthermia and sudden infant death syndrome: A systematic review. Pediatric Pulmonology, 48, pp. 1138-1145.

6. Boor, B.E., Järnström, H., Novoselac, A. and Xu, Y. (2014). Infant exposure to emissions of volatile organic compounds from crib mattresses. Environmental Science & Technology, 48, pp. 3541-3549.

7. Ayers, S., Creaser, C.S., Stockwell, P.B. and Cairns, E. (1997). Fungal volatilisation of antimony and the sudden infant death syndrome hypothesis. FEMS Microbiology Letters, 158, pp. 261-265.

8. Istituto Nazionale di Statistica (ISTAT). (2024). Cause di morte – Mortalità infantile 1970-2024. Rome: ISTAT.

9. Sørensen, H.B. and Andersen, S.K. (1998). National ban on polyurethane crib pads and subsequent decline in sudden infant death syndrome. Ugeskrift for Læger, 160, pp. 2634-2637.

10. Statistical Office of the Republic of Slovenia. (2024). Causes of death – Infants 2023. Ljubljana: SORS.

11. Moon, R.Y. and Hauck, F.R. (2016). Trends in sudden infant death syndrome: 2000-2015. Pediatrics, 138, e20162857.

12. Sprott, T.J. (2004). Cot death – Cause and prevention: Experiences in New Zealand 1995-2004. Journal of Nutritional & Environmental Medicine, 14, pp. 221-232.

13. Filiano, J.J. and Kinney, H.C. (1994). A perspective on neuropathologic findings in victims of the sudden infant death syndrome: The triple-risk model. Biology of the Neonate, 65, pp. 194-197.

14. Kinney, H.C., Richerson, G.B., Dymecki, S.M., Darnall, R.A. and Nattie, E.E. (2009). The brainstem and serotonin in the sudden infant death syndrome. Annual Review of Pathology, 4, pp. 517-550.

15. Schwartz, P.J., Stramba-Badiale, M., Crotti, L., Pedrazzini, M., Besana, A., Hullin, R., et al. (2018). Genetic cardiac channelopathies and sudden infant death syndrome. Heart Rhythm, 15, pp. 782-787.

16. Weese-Mayer, D.E., Rand, C.M., Ackerman, M.J., Bakewell, S.C., Detwiler, D.A., Kinney, H.C., et al. (2017). Autonomic dysregulation and sudden infant death syndrome. Pediatrics, 140, e20163703.

17. Poets, C.F., Schoeber, M., Wallis, S. and Kemp, J.S. (1999). Negative intrathoracic pressure and petechiae in an experimental asphyxia model. Pediatric Research, 46, pp. 81-85.

18. Brooks, J.H. and Burchfield, D.J. (1995). Hyperthermia increases pulmonary blood flow in neonatal swine. Pediatric Pulmonology, 19, pp. 78-84.

19. Hale, J.R., Aldridge, W.N. and Green, T. (1997). Neurotoxicity of stibine in neonatal rats. Toxicology Letters, 92, pp. 29-35.

20. Bettelheim, K.A. and Goldwater, P.N. (2004). Staphylococcus aureus α-toxin in sudden infant death syndrome. Journal of Medical Microbiology, 53, pp. 715-718.

21. Selvaggio, G.N. and Biondi, G. (2023). Cardiac conduction-system abnormalities in sudden unexpected infant death: A systematic review. Diagnostics, 13, p. 270.

22. Prandota, J. (2014). Hippocampal dentate gyrus maldevelopment in sudden infant death syndrome. Journal of Neuropathology & Experimental Neurology, 73, pp. 725-739.

23. Knight, H.J., Brown, L., Smith, D.L. and Richardson, B.A. (1993). Sudden infant death syndrome and the toxic-gas hypothesis. Letters in Applied Microbiology, 17, pp. 94-97.

24. Golding, J., Scott, A.M. and Lakhani, P. (1995). Concentrations of antimony in infants dying from sudden infant death syndrome and in other infants. Archives of Disease in Childhood, 73, pp. 284-287.

25. Simone, P., Williams, M.B. and Lubis, J. (2020). Identification of phthalate plasticisers and flame-retardants in crib-mattress components. Analytical Chemistry, 92, pp. 3006-3013.

Article sent: 27.12.2025