

Warning: controversial contents about SIDS and autism.

Disclosure: the views expressed here are those of myself only.

They challenge a “holy cow” ... a firmly held belief and dogma of my medical colleagues.

The “holy cow” that I am questioning concerns supine sleep for young infants.

The following is based on two publications, which have been through peer-review. The first was accepted almost immediately, an hypothesis for how supine sleep protects from SIDS.

The second was rejected by 5 high profile journals. In each instance, reviewers made thoughtful and helpful comments and wished me luck, in each instance editors very politely found some (seemingly spurious) reason to reject! The 6th editor obviously had more courage 😊 !

The first paper states that supine sleep prevents SIDS by acting as a stressor. The second paper argues that when the whole population experiences this stressor, the incidence of autism increases.

The two articles present one single coherent and cohesive argument: taking any piece of it in isolation and making an issue out of it is doing me and my argument a disservice, as well as potentially be a disservice to all newborns and our society.

My intention is not to make simplistic public health recommendations, but to provide the correct scientific information. This will allow parents to make an informed choice, and to guide scientists in more fruitful avenues of research.

What follows is a summarised layman’s version of the “story line” from both the articles. There are links to more details (I shall add more in future), and I refer to the actual articles for further details. There are no references, readers wanting such should read the full articles for the proper context. The two articles cover very complicated and difficult concepts, and in making this everyday language explanation some important detail gets left out. I also have to assume that you the reader have some prior knowledge of SIDS and autism, explaining every concept in greater detail would make this into a book.

Nils Bergman 160726

Sudden Infant Death Syndrome (SIDS) is every parent’s nightmare, a terrible tragedy. It is not only terrible for parents, but for those carers and health professionals that support them. It is altogether an extremely emotional issue. What I will explain may evoke emotional reactions of many kinds. However, in order to properly understand the subject and improve our understanding for better care in the future, I am setting aside my own and your emotional mindset, and looking at this whole question in an objective and scientific way.

Sudden Infant Death Syndrome (**SIDS**) is best explained in the [Triple Risk Model](#), for which Hannah Kinney states that there is “always” a defect, in 70% of cases that defect is affecting a specific breathing centre involved in autoresuscitation.

So how do we understand SIDS? Clinically it is characterised by repeated episodes of low heart rate, low blood pressure, cessation of breathing, a kind of collapse. Recovery starts with breathing, then

the heart rate picks up. These episodes are sometimes observed, but more often not as they occur during sleep. Eventually there is a failure of recovery, and a “sudden death”. Going back a step, the “collapse” state can be traced to the activity of a specific brain centre (vIPAG, ventro-lateral Peri Aqueductal Gray). This is a primitive part of the brain that we inherited from reptiles. Primitive reptiles only have parasympathetic systems, and the defence of slowing down works for them. The vIPAG orchestrates a primitive reptilian defence mechanism, acting on several lower brainstem centres, which are purely parasympathetic, slowing everything down by the action of the vagal nerve. Reptiles are cold-blooded and have slow brains, warm-blooded big brained mammals cannot survive for long in this way. When a mammal experiences the unopposed activity of the vIPAG, it is in fact an autonomic nervous system collapse. That can be useful as a temporary measure (as in a faint), but only very temporary. A profoundly powerful autoresuscitation mechanism kicks in, beginning with a strong gasp (details in first article). It has to be strong enough to push oxygen to the arteries that supply oxygen to the heart muscle. If the heart is forced to beat and there is no oxygen, it kills the heart muscle cells. So when oxygen arrives, the heart starts to beat.

I have labelled the pure vIPAG discharge as an Adverse Autonomic Event (AAE).

In SIDS victims, the gasp mechanism is defective. The gasp mechanism is extremely sensitive to nicotine, even in healthy subjects nicotine makes the gasp weaker, but much more so in those with a defect. For babies with the defect, it is nicotine that is deadly. (Not prone sleep, not skin-to-skin contact). The defect comes with degrees of severity, which might explain why even under the best of circumstances, babies with a severe defect succumb.

What triggers the discharges from the vIPAG that cause episodes of autonomic collapse ? The vIPAG is part of the neural circuitry for defence. Several parts of the brain contribute to neuroception or “threat appraisal”, but the strongest signals to the vIPAG come from the amygdala, which is the emotional brain. The brain is not off or resting during sleep, it is actually extremely busy. This busyness is seen in REM (Rapid Eye Movement) sleep, during which the body (soma) is disconnected from the brain (atonia), but only below the eye muscles! It is believed that negative emotional processing is taking place, and the atonia prevents negative acting out. However, the emotional responses and their effect on the autonomic nervous system are still very active, and occasionally the vIPAG will fire to produce the AAE. This arises from a deep neurological activity that tells the sleeping brain that there is a threat, or a deep sense of being “unsafe”. In large sleep studies (CHIME) these AAEs were in fact not uncommon, and the autoresuscitation was seen working well.

However, in subjects later dying of SIDS, the autoresuscitation was evidently weaker. Further, SIDS has been shown to be more common during REM sleep.

So where does supine sleep – “back to sleep” - come in?

Davies observed in [Hong Kong](#) that SIDS was rare among the Chinese who kept their infants sleeping on their backs. This eventually led to “back to sleep” campaigns in many countries, with dramatic lowering of SIDS cases. Many countries and paediatric associations give strong advice that all young infants should sleep on their backs (supine sleep).

Nobody has however been able to explain the mechanism for this decreased incidence of SIDS. Much research funding is currently being spent on understanding why prone sleep is harmful, on looking for the mechanism by which prone sleep contributes to SIDS.

Disclosure: the above is not controversial.

What follows is controversial.

When looking closely and objectively at sleep, it is clear to me from the biology and the clinical evidence that prone sleep is the biological norm.

In contrast, supine sleep is measurably stressful, for example using heart rate variability. In premature infants, the stressful effect of supine sleep is even more dramatic in the clinical context. I describe this in more detail in the first article.

Supine sleep is a stressor. I make this statement, supported by research and clinical evidence. Prone sleep is in fact the normal healthy sleep for all human beings, from neonates to adults.

This insight does in fact explain how supine sleep lowers the incidence of SIDS.

Supine sleep is a stressor. Most people do not know that the parasympathetic system actually deals with stress as much as the sympathetic system does. But in the context of supine sleep it is in fact the sympathetic nervous system that is activated. There are three other parts of the Peri Aqueductal Gray, and these we call “higher” or more advanced parts, and they all use nuanced combinations of sympathetic and parasympathetic. This prevents the vPAG from firing its pure parasympathetic output. When these PAG parts are active, they prevent the autonomic collapse (the AAEs) from happening. This is one way that supine sleep protects from SIDS.

There is a second way also. The hormone (neurotransmitter) involved in the breathing defect is serotonin, but this hormone is also involved in control of sleep. With the defect, it is likely that REM sleep is increased. Additionally serotonin is involved in anxiety, which may also increase negative emotion that needs even more REM to process. Supine sleep changes sleep architecture, basically by raising the “state arousal”, babies sleep less deeply, and therefore they have less REM, and so less risk of AAE, and so less risk of SIDS. What it also means is that babies do not actually sleep as well when on their backs.

These are two stressor mechanisms that explain how supine sleeps reduces SIDS incidence.

Many parents actually know that babies do not sleep as well on their backs as on their stomachs ... it is clear from knowing and observing their infant. Nursing staff, especially those that spend many hours actually watching over and caring for preterm infants and small newborns, also see and know this. Infants simply do not sleep properly, or well, on their backs. And as soon as the supine baby has enough motor development (usually between 3 – 6 months), it will turn itself to sleep on its front.

Researchers looking for harm in prone sleep compare prone and supine, see that “state organisation” in supine is higher than prone, and therefore assume that the harmful prone is “too

deep". I argue that the supine sleep is "too light", basically poorer quality sleep with less sleep cycling. Babies do not die from being in "too deep sleep" ... I doubt there is a such thing in the physiological state. They can be drugged or medicated into such a state, or have some other pathology, but that has nothing to do with SIDS.

And so I believe that this proposal that supine sleep is a stressor that prevents autonomic collapse (AAE) is the most biologically plausible mechanism that explains the mechanism of protection of supine sleep on the lowered incidence of SIDS.

(Summary of first paper: "Proposal for mechanisms of protection of supine sleep against sudden infant death syndrome: an integrated mechanism review."

Supine sleep DOES DECREASE SIDS in the population.

If we changed this, if we made the recommendation that all infants should sleep on their fronts or stomach, there would be more SIDS cases.

There is very good [epidemiological](#) evidence that supine sleep campaigns have been successful in reducing the incidence of SIDS. It was reduced by 50% in the USA between 1994 and 2000.

In Sweden it was reduced by 70 – 80%! Epidemiology only makes observations: it cannot be used as evidence to explain or prove anything. Two other epidemiological changes took place in Sweden during this same time that might have made the additional lowering effect: breastfeeding rates increased, and smoking rates decreased dramatically, augmented by the increased use of chewing tobacco (snus). This epidemiology suggests to us (again) that nicotine is the key culprit.

So let us look more closely at some snippets from the science of [epidemiology](#).

SIDS is defined as a rare disease: anything that happens less than 1 per 1000 of the population. It was always a rare disease, and now it has become a bit "more rare". "Rare disease epidemiology" takes into account some matters that have been ignored in the current debate.

For example, the [Triple Risk Model](#) states that there must be a defect.

The question then should be "how common is that defect" ?

In very detailed research, a defect (usually in the brainstem) has almost always been found in SIDS cases. In control subjects (died of something else), such defects have never yet been found. It is technically very difficult to find that defect, and so we do not know the incidence (number of babies born per year per population) of the defect. However, it is likely to be low, or even very low.

Some defects are relatively harmless (0% in terms of mortality), and some can be fatal (100%). So what is the "fatality rate" of the defects involved in SIDS? Again, we do not know. But since they have never been found in controls, it is likely that the fatality rate is high. We could make some calculated guesses, based on what we have seen in response to supine sleep campaigns. SIDS incidence we know approximately, supine sleep effect we know approximately from the "resulting

incidence". So what used to be 10 babies of every 10 000 dying, the effect size of 50% reduction means that only 5 babies of 10 000 are dying.

If defect was 1000 per 10000 (10% of population), it would have shown up in controls. The more controls we have with no defect, the smaller the defect incidence has to be. I make the conclusion that the defect is a rare finding in the population, closer to 20 or perhaps 40 per 10 000. Further, it is a "nasty" defect with a high fatality. But not absolute, it can be impacted by an intervention or treatment that is powerful enough to counteract it; in this case supine sleep. Nicotine reduction might have a similar effect size.

That "all babies must sleep supine" is in many places an aggressive public health slogan. And superficially, this makes very good sense. But the "very good sense" is actually based only on epidemiology, and then on an insufficient understanding of epidemiology.

The effect size as measured in the population also depends on the proportion of the population that complies, that actually follow the advice to put their babies on their backs to sleep. At times and in some places the public health campaigns are emotionally forceful. Now if the intervention that is being "forced" on parents and children is totally harmless and has no adverse effects of any kind, then all is well. When providing a treatment (or prevention) against a rare disease that must be taken by the whole population, then we must also be aware of all possible rare side-effects that may appear in the population. Are such side-effects common, as rare as the rare disease, or very rare; and are those side-effects mild so that we can accept them, or are they severe that we have to consider very carefully what we are doing?

There is very little information provided on any possible negative consequences. And even if the intervention is not totally harmless, but still mild in comparison to thousands of SIDS deaths every year, then we should happily support the supine sleep message.

BUT

WHAT if ...

... what if supine sleep is not harmless?

... what if supine sleep is even harmful?

I do not believe there has been an adequate debate, discussion or discourse, much less provision of epidemiological and scientific data, on which a proper benefit and risk assessment can be made.

I call myself a "public health physician", and I believe I am acting responsibly in examining this issue in greater detail. So I will repeat:

Supine sleep DOES DECREASE SIDS in the population.

If we changed this, if we made the recommendation that all infants should sleep on their fronts or stomach, there would be more SIDS cases.

WHAT if ...

... what if supine sleep is not harmless?

... what if supine sleep is even harmful?

Sleeping on one's stomach (prone) is biologically normal. Almost all animals have a "righting reflex" that keeps their backs upwards, hence prone. No animals sleep supine (excepts bats, sloths and the odd domestic cats and dogs), and it has been shown that for adult humans prone sleep is healthier.

The research on benefits and risks of prone and supine sleep is totally clear when it comes to low birth weight and preterm infants. Such neonates when they sleep on their backs (supine) have worse oxygenation, have worse sleep, have more crying, and have more arousals during sleep which prevent healthy sleep cycling. Sleep cycling is actually when the brain wiring takes place, the key mechanism underlying healthy neurodevelopment. (More in first article)

I make the statement again: Prone sleep is NOT harmful.

Having a brain stem defect exposed to nicotine is harmful.

It is only in the context of a brainstem defect – which is rare in the population – that supine sleep confers a benefit.

There are a number of harms already identified from supine sleep.

Plagiocephaly – a misformed and assymetrical skull – occurs in 1 of 60 babies that sleep supine. For most it is purely cosmetic, but in some cases it is correlated with delayed development, and in a few cases corrective surgery is necessary. At a public health level, parents may well accept this relatively minor malady for the trade-off that their own child did not die of SIDS. Even for those with an altruistic or epidemiological mindset, their own baby is paying a small price for a terrible price somebody else in the city would otherwise have to pay.

Supine sleep also causes delayed motor development. This is in fact quite substantial, and lasts into the second year. What is more, it affects the majority of infants who sleep supine, to a greater or lesser degree. Still, that is preferable to tragic deaths, even if those are rare (1 in 1000). The public health communication on this has however been bordering on dishonest. One line of response is to say that all babies should have "[tummy time](#)", almost a structured treatment, tacitly acknowledging that it is a preventive treatment to correct a harmful side-effect of another preventive treatment. A second line of response is more dissembling, which is to re-define this delayed motor development as the "new normal", arguing that it is only temporary and does not matter at all, so it can be happily accepted. Saving those lives is much more important.

My argument is that this is dishonest, because full disclosure of all information, and the sums and figures and resulting costs that make up this benefit risk assessment are not divulged to parents. And the possibility that any other harms are possible in the population is not entertained.

I do agree that saving lives is important. And I have provided a science based explanation as to how supine sleep can save the life of a baby with a defect in auto-resuscitation.

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So what could possibly alter the equation of benefit and risk for supine sleep?

I have argued: Supine sleep is a stressor.

I have also described that it has an effect size of 50% ... in research contexts, this is a very powerful intervention. But it exerts its very powerful effect in very few subjects ... since we cannot identify those subjects in the population, we provide the intervention to the whole population.

If supine sleep is a powerful intervention, is it also a “powerful stressor”?

It is sufficiently powerful to cause neurodevelopmental delay in the motor domain. What other domains might it affect, that we have not looked at?

Stressors impact development in several ways. Development occurs in sequence, according to a time table. Lower circuits are laid down first, and higher circuits build on lower circuits. The lower circuits are therefore more important, or critical. Once laid down, they are robust, but during the actual laying down they are sensitive to influences. In the better of circumstances, this sensitivity comes from the gene being awakened at the right time (expressed) and reading the environment in order to fine tune how it should express the protein it is programmed for. Epigenes are switches that can be altered by the environment, and subtly change how the gene makes its protein. This is called adaptation, and is healthy and good. When circumstances are unexpected and the environment is harsh, the adaptation process accelerates, making more extreme changes. This is not harmful in the short term, it is necessary to function in a harsh and hostile environment, but it comes with a trade-off that it cannot function in a benign world, it is now maladapted. Such mal-adaptation also comes with something called “increased allostatic load”, by which the whole organism runs at higher revs, with greater “wear and tear”, resulting in poorer physical and psychological health in future years, and a decreased life span. DOHaD (Developmental Origins of Health and Disease) is whole new science that has arisen from our understanding of these mechanisms. Epigenetics is a key to this science, and “methylation” refers to one specific epigene that is usually involved in switching off a specific receptor for a hormone or neurotransmitter, and the function of that hormone system can be fine-tuned by the degree of methylation. Stress comes with cortisol, which is one mediator of methylation. The more stress, the more cortisol, the more methylation, the more a system can be tuned down or out. The less responsive or active any system is, the less adaptable, or more maladapted.

Parallel to the above, supine sleep has a second major impact on development, and specifically neurodevelopment, which is to disrupt and decrease “sleep cycling”. It is during sleep cycling that neural circuits are laid down, or wired. There are several forms or stages or phases of sleep that the brain cycles through, all necessary for quality sleep. I have mentioned REM sleep earlier. REM sleep

is a higher level of state organisation, and comes in at least two forms, (tonic and clonic). Then there are several other intermediate phases of sleep until we get to the deepest level of sleep in a healthy sleep cycle, which is called “slow wave sleep” or Quiet Sleep. Very specific brain wiring steps take place in this phase, the consolidation of neuron to neuron connections. At the end of the sleep cycle, just before REM comes back, neuron to neuron connections are linked to circuits and networks of several circuits. Our brains support networks, and the sum of all networks is called the [connectome](#), and it is that sum which makes us: “we are our brains”. Again, this network connecting is happening according to a timetable in the baby’s brain, each circuit must be laid down in the right sequence for a higher circuit to build another network on it. When sleep is disrupted during a particular time, it is the circuits being laid down at that particular time that are sensitive and that can be disrupted.

Supine sleep is a stressor (able to switch off receptors) , and it disrupts sleep cycling (able to disrupt circuits). We are applying supine sleep to the whole population to successfully decrease the number of babies dying from SIDS. We do this during a “sensitive period” (as per triple Risk Model), during which the defect is known to exert its effect, with increasing age some other mechanisms appear able to counteract the defect, which presumably never goes away, but no longer causes death.

What circuits and networks are being laid down during the first three months of life, that could be adversely impacted by supine sleep? Some examples :

The amygdala to orbito-frontal lobe connection (emotional brain to social brain)

The amygdala to medial prefrontal connection (emotional brain to external approach orientation)

Frontal lobe to fusiform gyrus connection (social brain to face recognition area ,eye to eye contact)

Dopamine reward networks connecting to oxytocin networks (sociality becomes rewarding).

Saliency switch network is part of the threat appraisal (tune mind inwards or outwards)

Default mode network is the inwards system when appraisal says external attention not required.

The above circuits - that may be potentially disrupted by supine sleep – are implicated in autism.

Autism is properly referred to as “Autism Spectrum Disorder” (ASD), and has recently been redefined. It is now also best understood as a “connectome disease”, which means that it is a failure of several networks within the connectome to function properly together. The networks that are implicated include the ones that I have listed above, and some others. Simplistically, the concept of the autism “spectrum” reflects that a whole variety of disease expression is caused by the variety of different ways the networks internally may be more or less disrupted, and the ways the network connections to other networks may be more or less disrupted.

Autism is also a rare disease. At least, it was a rare disease some 40 years ago, now it is occurring at an incidence more than 1/1000.

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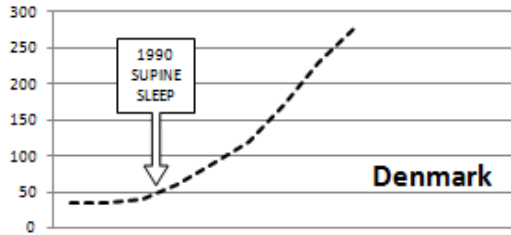
BUT – could it be that the price we pay for less SIDS is more autism?

It was epidemiological evidence that led to our discovery that supine sleep decreased the incidence of SIDS.

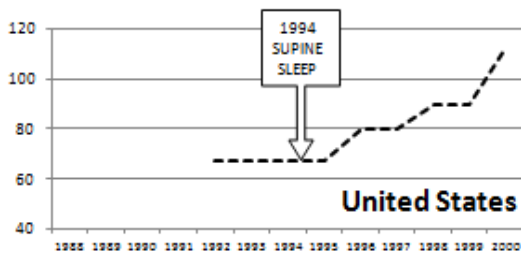
If what I have described above is possibly true, that supine sleep could increase the incidence of autism, then there should be epidemiological evidence to support that also.

Finding such evidence is very difficult. Autism was a rare disease, and data from more than 40 years ago was not reliable, purely because rare disease data is very hard to get. But then autism became more common and we got lots more data. But then it became more popular, and cases that were maybe not autism got called autism. And then it became redefined, and more cases might suddenly be called autism that were not before. Or vice versa. And then it turns out that actually more people had some of the symptoms of autism than we realised, and that maybe it was always quite common. Much confusion. There is a great deal of literature discussing this, and very little providing data. I have searched hard for decent timeline data on autism incidence, and have found such from only five countries.

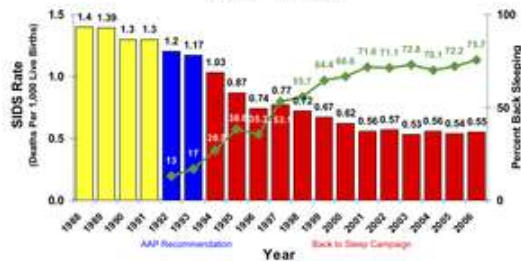
Let us start with Denmark, which is also appropriate as it has to do with a major scandal related to autism. Wakefield is well-known for suggesting that measles vaccine was the cause of the increased autism in the world. It was in fact in Denmark that research was done that proved he was wrong. Denmark is a small country in which everybody belongs to the same health system with standardised diagnostic information stored in robust servers. Very consciously autism incidence was measured at the same time as measles vaccine was given, and then in 1992 the vaccine was changed to remove the mercury that was being blamed. But careful prospective recording showed that autism increased starting in 1991, and continued increasing thereafter. Measles vaccine does not cause autism. But what this very carefully collected data does show is a “change-point”, a specific year when the autism suddenly started increasing – 1991. This was also the year that the supine sleep campaigns were started in Denmark and all of Scandinavia, with a dramatic lowering of SIDS cases.



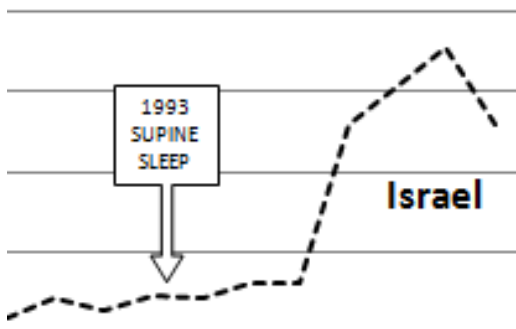
Supine sleep campaigns can be dated and therefore plotted on a timeline graph. Actually one would prefer to plot the compliance to the campaign, by measuring the percentage of the population that did sleep supine. Such data is also very scarce. There is such available from the USA, with the reality check that the large sample got from phone interviews showed much higher compliance than the small sample when sleeping position was actually checked properly (on the phone parents reported what they knew was the right answer, not what they were doing !) In the USA, the first advice on supine sleep came in 1992, the campaign started in 1994. The most accurate autism data I regard as coming from the ADDM, and it shows the inflection point for autism at 1994.



SIDS Rate and Back Sleeping (1988 – 2006)



SIDS Rate Source: CDC, National Center for Health Statistics.
Sleep Position Data: NICHD, National Infant Sleep Position Study.

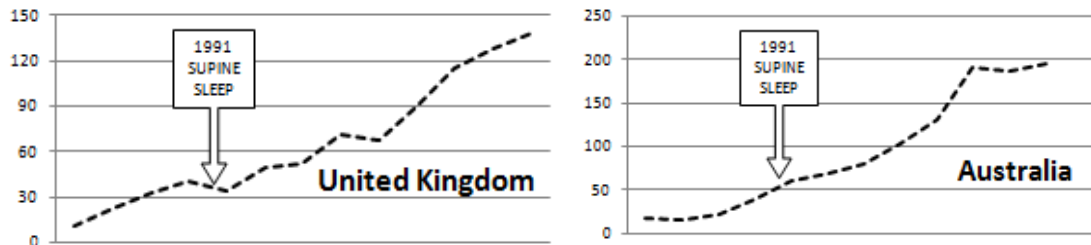


In Israel, the supine campaign started in 1993.

Israel is interesting also, as the data I can find comes from insurance claim registry. There was a sudden spike of claims for diagnosed autism in 1996, and there had not been a change in definitions or system or monetary incentive around that time. The usual autism incidence definition is based on the population of 8 year olds, because it can take up to 8 years for autism to show itself. But this was insurance claims, and the age of the claimants was 39 months, meaning they were born

“Once is happenstance, twice is coincidence, three times is enemy action” Ian Fleming

I have similar data also for the UK and for Australia, and along with Israel these show another phenomenon: the incidence of autism rises and then levels off. There is a reason for this also: to get autism is actually not easy. Firstly you need to have susceptible genes, and these occur in less than 10% of population, and are found in families. If you do not have these genes, you will not get autism. Just like SIDS, if you do not have those defects, you will not get SIDS.



Now when the whole population is sleeping supine, only that proportion that have sensitive genes, and that sleep supine, and almost certainly that also have other stressors that came at just the wrong point in the developmental timetable, only that proportion will be affected. 90% of the population will not get autism, because they do not have the susceptibility, (they might of course get other problems.) But to get autism, you need to have several networks in the connectome susceptible and affected.

Supine sleep is a powerful intervention, with a powerful stressor effect, that is likely to have increased the incidence of autism.

This increase in autism occurred only in the decade following the campaigns, in countries with stable supine sleep rates, any increases in autism requires another explanation. The commonest is called secular trend: autism is not increasing but being increasingly recognised or diagnosed. But it is also quite possible that one or more other environmental factors, over and above supine sleep, is operating to increase autism incidence.

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BUT – could it be that the price we pay for less SIDS is more autism?

I am repeating this !!

I have presented an hypothesis, which can also be called a research question.

Does supine sleep increase the expression of autistic criteria in the population?

I am not recommending that all infants should sleep on their fronts (prone).

What am I recommending?

Any hypothesis – and this one - is primarily aimed at the research community. I am proposing that research into the harmful effects of prone sleep are fruitless and wasted. I am advising that we should more keenly focus on the potential harmful effects of supine sleep, so that we can make a more responsible weighing of benefit and risk in the population as a whole.

The public health community should particularly apply themselves to this hypothesis. We should find the answers as to defect prevalence for SIDS, and accurate incidence of autism as a diagnosis but also with respect to distinct criteria and their underlying biology. The epidemiological link between supine sleep and autism could be verified or refuted, and additional factors identified.

This will allow better focus for basic science and for clinical research. With respect to SIDS, infants with a defect should certainly sleep supine, but how do we identify them? And when we can identify them, we can do much more than just provide supine sleep. With respect to autism, a broader understanding of the connectome and early childhood development may have benefits for society as a whole.

The end result should be a clear and accurate assessment of what are the actual, weighted risks of different choices, which can better advise public health policy, and practical parenting advice. The current public health policy (requiring unquestioning adherence to supine sleep) fails to do this.

Some of you reading this are parents (some of you are also researchers).

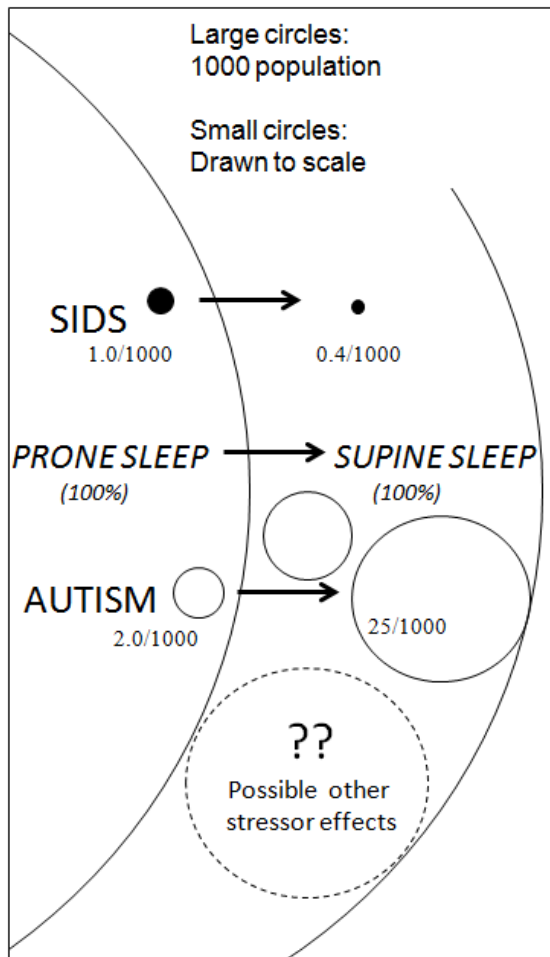
Some of you read this with the grief and loss of having experienced SIDS. And others with as intense but very different grief and loss living with the ongoing reality of autism. To both groups I would start by saying “Don’t blame yourselves”. The SIDS defect is very rare and very deadly. The autism defects are very many and pervasive, and some have already started expressing in the womb. Blame and guilt are not applicable. But tend your grief and your sorrow, don’t bottle them up, do not go it alone, and if necessary seek help.

Some of you are prospective parents, and some have young babies right now. You want to make wise choices right now. What you are reading here makes those choices extremely difficult. For others of you the choice is not difficult, you already know that experiencing SIDS would be too intolerable, it is a small risk to deal with autism. For others you may already see autism as the worse thing, and see the very small risk of SIDS as acceptable.

If you are reading this you are most likely adult enough to understand, smart enough to want to understand, and wise enough to make up your own mind. To such a mindset, I am providing information as factually as I am able: supine sleep certainly decreases SIDS, but is likely also to increase autism. Both are rare. But if your family has autism, or are vaguely asocial, you may want to put extra effort to providing the environment that decreases risk of autism expression, and sleep position may be a part of such. If your infant has shown the slightest sign of early apnoea or sign of

autonomic instability, you may want to emphasise supine sleep in case this was because of a brainstem defect. Perhaps there might be ways of practising supine sleep and doing compensatory measures to decrease risk for autism.

It might be helpful to consider the following graphic to grasp the issue of how rare SIDS and autism are, and a perspective on the impact of supine sleep.



The large circle that doesn't fit into the diagram on the left represents the whole population, or let us say 1000 babies. And in this circle all babies are sleeping prone, on their stomachs.

SIDS occurs in 1 of those 1000, the large black circle labelled SIDS. I have drawn this to scale, 999 babies of a 1000 do not get SIDS.

In this circle of prone sleeping babies, the white circle represents those who get autism. The black circle is pretty accurate, but data for white circle (autism) is very hard to come by. From a detailed review, before 1990 it was probably the same as SIDS, but since it may not have been recognised I have made the circle twice as big.

To the right is the circle in which the whole population is sleeping on their backs, supine. SIDS and autism are again drawn to scale.

In the second article I argue that a fourfold increase may be due to supine sleep, the circle above the arrow. The larger circle represents 'social inhibition disorders', and the dotted circle other possible adverse outcomes.

When you have carefully weighed the options, and applied your mind to all the available information, you should follow your heart. Note that "all the available information" does include that which very strongly and passionately disagrees with what you have read from me: please read that also. Whatever adversity that then befalls, accept as the outcome of a higher source. You made a choice as best as you were able, you have no control over the genes you got from your parents, nor any other vagaries of fate.

Delete "guilt" – which is being used as a lever by authorities to extract blind obedience. Only when you have knowingly done something you knew was not the best thing to do, only then are you guilty.

In conclusion, this is difficult territory. Very many things in life can be seen as a benefit risk trade-off. When this affects life (as in autism) and death (as in SIDS), the choices become extremely difficult. Ignorance is however no excuse, we need to look for the true facts.

Supine sleep DOES DECREASE SIDS in the population.

If we changed this, if we made the recommendation that all infants should sleep on their fronts or stomach, there would be more SIDS cases.

BUT – could it be that the price we pay for less SIDS is more autism?

Some background

I have for ten years been researching the neuroscience that underlies skin-to-skin contact, or what is popularly known as KMC, Kangaroo Mother Care. This was first introduced to English speaking doctors as the “myth of marsupial care”. As a result this was originally seen as something of an alternative medicine and not of real importance or value. My research provides the biological rationale for skin-to-skin contact as being the essential environment for neonate primates including the human primate, that critically supports all aspects of reproduction and development. The summary of this I have made into a position statement [on this website](#).

As skin-to-skin contact has become more widely accepted and practised internationally, reports of infants dying on their mothers’ chests are being reported, both in hospital and in homes after discharge. In any grief crisis, a basic human emotional response is blame, and it is still common that skin-to-skin is blamed for the death. Being confronted with this blame (whether it is emotional and irrational or not), I applied my mind to the facts and the neuroscience of such deaths, which resulted in these publications ,and this article.

SIDS has very specific definition, and is often confused with other forms of unexpected deaths in neonates and infants. Sudden Unexplained Postnatal Collapse (SUPC) occurs in infants below 7 days of age, SIDS by definition only occurs in infants older than 28 days.

Skin-to-skin contact must be practised with safe technique. When done badly or incorrectly, it is a possible cause of SUPC, but not of SIDS. ASSB (accidental suffocation and strangulation in bed) can occur at any age, and can be a consequence of bad skin-to-skin technique. It can also be a consequence of bad incubator technique, as well as unsafe sleep environment.