SUPPLEMENTARY MATERIAL

Search

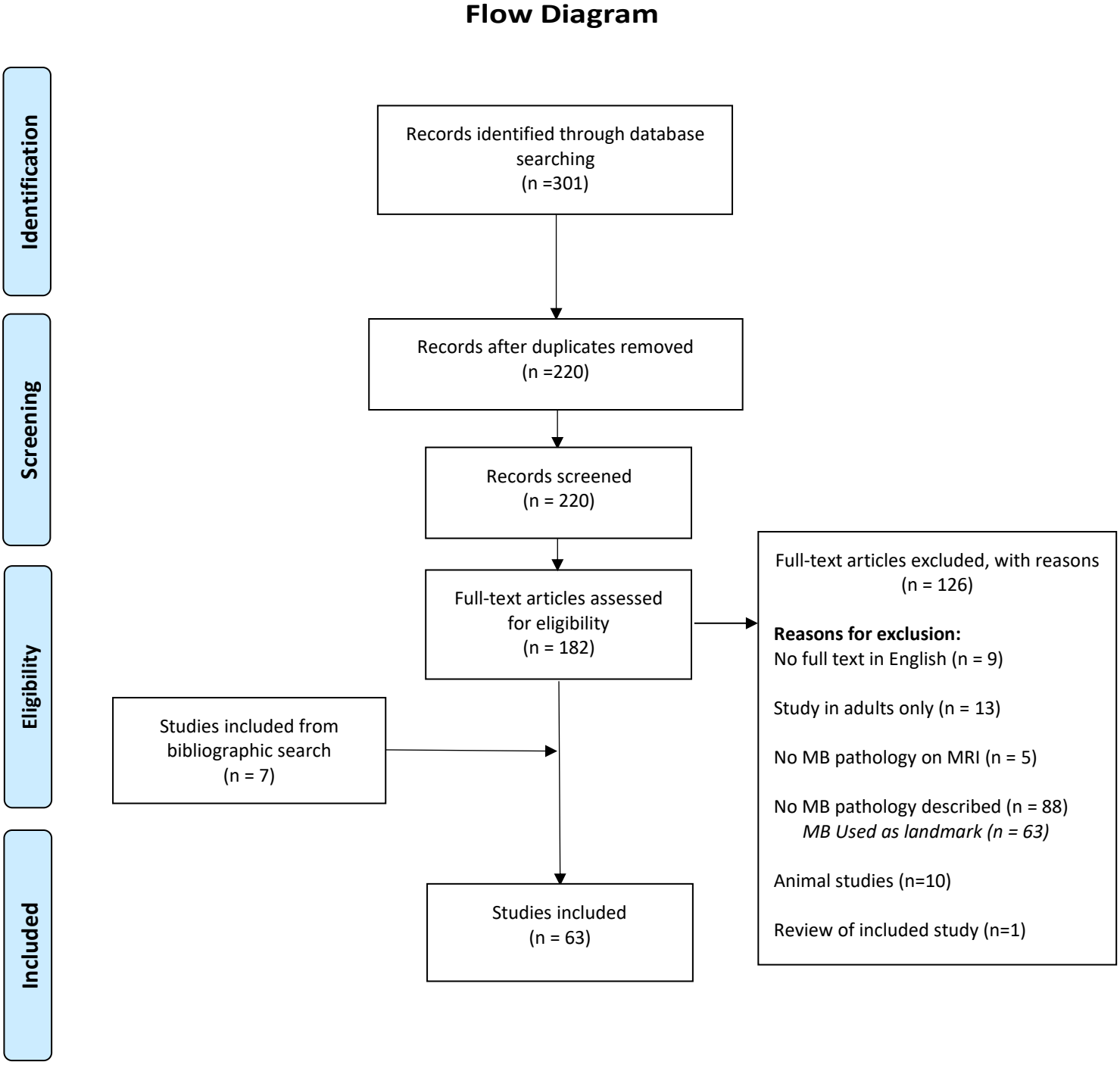
Last search performed on the 1th of October 2021.

Pubmed:
(((mammillary bodies) OR (mammillary body)) AND ((pediatric) OR (child) OR (children) OR (infant) OR (neonatal) OR (adolescent)) AND ((MRI) OR (radiology) OR (T2) OR (DWI)))

Alternative spelling and variation of the words are automatically included by the pubmed search engine

Embase:
('mammillary bodies'/exp OR 'mammillary bodies' OR (mammillary AND bodies) OR 'mammillary body'/exp OR 'mammillary body' OR (mammillary AND ('body'/exp OR body))) AND ('pediatric'/exp OR pediatric OR 'child'/exp OR child OR 'children'/exp OR children OR 'infant'/exp OR infant OR neonatal OR 'adolescent'/exp OR adolescent) AND ('mri'/exp OR mri OR 'radiology'/exp OR radiology OR t2 OR dwi).

Flow-diagram showing the search and assessment of studies included



Overview of included articles arranged by underlying condition and age of (youngest) patient

Author Year	**Study design**	**Number of participants**	**Age of participants**	**Condition**	**Type of MB pathology on MRI** *Other relevant outcomes (?)*	**Lowest slice thickness used**
Wani et al. (2016)[1]	RS	22 patients	45 days – 8 months	Thiamine deficiency (WE)	Hyperintense signal on T2 weighted imaging in 1	4 mm. Interslice gap: 1 mm
Kornreich et al. (2005)[2]	RS	6 patients	2-10 months	Thiamine deficiency	T2-weighted images showed hyperintensity of the MBs in 5 patients On follow up: atrophy of the MBs in three patients	Unknown
Fattal-Valevski et al. (2005)[3]	RS	9 patients	2.5-12 months	Thiamine deficiency (WE)	T2 hyperintense MBs in 1 patient MRI findings improved 5 weeks later	Unknown
Gliebus et al. (2014)[4]	CR	1 patient	19 months	Thiamine deficiency (WE)	Restriction of diffusion in MBs with increased signal onT2-weighted and FLAIR images Follow up 1.5 months later, showed a marked improvement of the signal changes	Unknown
Oka et al. (2001)[5]	CR	1 patient	3	Thiamine deficiency (WE)	T2 hyperintense MBs Follow-up MR examination after 1 month demonstrated complete resolution	Unknown
Darlington et al. (2015)[6]	CR	1 patient	5	Thiamine deficiency (WE)	Bilateral enhancement MBs	Unknown
Zuccoli et al. (2007)[7]	RS	26 patients 13 no alcohol abuse (NA)	6-81 (mean 46.6 ± 19)	Thiamine deficiency (WE)	Symmetric lesions in MBs in 58% (in NA patients 38%) *Contrast enhancement of MB statistically positively correlated with alcohol abuse group*	Unknown
Zuccoli et al (2009)[8]	RS	56 patients	6 – 88 (50.3 ± 17)	Thiamine deficiency (WE)	Evidence of symmetric lesions in the MBs (45%) *MB contrast enhancement was significantly associated with alcohol abuse*	Unknown
Srivastava et al. (2012)[9]	PS	11 patients 8 controls	7.8 ± 2.7 8.8 ± 2.5	Thiamine deficiency	MBs significantly smaller in patients At follow up MB volume significantly improved	3 mm
Vasconcelos et al. (1999)[10]	Review of CRs	31 patients (8 with MRI)	11 ± 6.5	Thiamine deficiency (WE)	Increased signal in T2-weighted images and contrast enhancement of MB in all 8	Unknown

Harter et al. (1995)[11]	CR	1 patient	11	Thiamine deficiency (WE)	MB enhancement on postcontrast examination	Unknown
Cooke et al. (2006)[12]	CR	1 patient	11	Thiamine deficiency	Typical midline increased signal intensity in the dorsal mid-brain, thalami, and MBs on FLAIR imaging	Unknown
Sparacia et al. (1999)[13]	CR	1 patient	12	Thiamine deficiency (WE)	Intense enhancement of the MBs Follow-up 1 month later: no signal abnormalities were found nor was there MB atrophy	Unknown
Gupta et al. (2012)[14]	PS	10 patients 11 controls	15 ± 11 40 ± 12	Thiamine deficiency	MB volume changes are primarily a consequence of thiamine deficiency, which may secondarily result in microstructural changes in the fornix	1 mm
Lamdhade et al. (2013) [15]	CR	1 patient	16	Thiamine deficiency	Gadolinium enhancement of MBs and vermis	Unknown
Liu et al. (2006)[16]	CR	1 patient	16	Thiamine deficiency (WE)	Gadolinium enhancement on day 5 in the right MB, this had not been found on the initial MRIs. *PA: focal hemorrhage, edema and hypertrophy of the endothelial cells of the right MB.*	Unknown
Arana-Guajardo et al. (2012)[17]	CR	1 patient	17	Thiamine deficiency (WE)	Hyperintense signals in MBs on T2 FLAIR images	Unknown
Samanta et al. (2015)[18]	CR	1 patient	17	Thiamine deficiency (WE)	abnormal T2 prolongation and restrictive diffusivity of bilateral MBs	Unknown
Renthal et al. (2014)[19]	CR	1 patient	'adolescent'	Thiamine deficiency	Symmetric abnormal T2 prolongation of the mammillary bodies	Unknown
Lyons et al. (2016)[20]	CR	1 patient	18	Thiamine deficiency (WE)	symmetric signal T2 hyperintensity and restricted diffusion in het MBs	Unknown
Khalsa et al. (2016)[21]	RS	32 patients 30 controls	19.4 ± 7.3	Thiamine deficiency	Significantly smaller MB volumes in underweight group. Weight-restored group exhibited significantly larger MB volumes.	Oversampled to a resolution of 0.2 x 0.2 x 0.2 mm3
Fei et al. (2008)[22]	RS	12 patients	43.9 (16-69)	Thiamine deficiency (WE)	Gadolinium enhancement of MB in 2 of 3 patients. No atrophy of MB	Unknown

Zuccoli et al. (2010)[23]	R	23 patients in 17 articles	Unknown	Thiamine deficiency (WE)	16/23 cases showed bilateral alterations in the thalamus (70%), 13 in the periaqueductal region (57%), 11 in the MBs (47%) and three in the tectal plate (13%).	Unknown
Lallas et al. (2014)[24]	R	Unknown	Unknown	Thiamine deficiency (WE)	Common findings include symmetric T2 hyperintensities in dorsal medial thalamus, MBs, periaqueductal gray matter, and tectal plate.	Unknown
Zuccoli et al. (2009)[25]	R	40 patients In 16 articles	Unknown (Pediatric and adult cases)	Thiamine deficiency (WE)	Contrast enhancement of the MB may be the only sign of WE	Unknown
Annink et al. (2021)[26]	RS	50 (22 HT and 28 non-HT)	3-8 days + FU 10 years	HIE	At 10 years of age, MB atrophy was present in 17% in the non-HT group and 50% in the HT group	0.8 mm
Molavi et al. (2019)[27]	RS	235	3-10 days	HIE	abnormal high signal on T2-weighted sequences (13.2%) restricted diffusion (2,5%) atrophy at FU (89%) (in 9 patients)	2 mm
Lequin et al. (2021)[28]	RS	231	< 2 weeks	HIE	abnormal T2 signal intensity with swelling (41%) atrophy at FU (44%) (in 18 patients with abnormal MBs)	Netherlands 1 mm Genoa 2-3 mm
Bindschaedler et al. (2011)[29]	CR	1	8	HIE	Severe bilateral sclerosis of the hippocampal formation as well as atrophy of the fornix and MBs	1.2 mm
Geva et al. (2020)[30]	DS	20 patients 17 controls	14.05 ± 3.86 16.24 ± 7.05	HIE	MB small in 50% of patients *Movement coordination deficit affecting the hand and the wrist in patients exposed to early hypoxic-ischaemic brain injury may be related to reduced volumes of the caudate nucleus*	1 mm
Dzieciol et al. (2017)[31]	RS	18 DA 18 controls	20 (11-35) 19 (10-35)	HIE	Atrophy in DA group (67%) No Atrophy in controls (0%) *Associated with memory loss*	1 mm
Kumar et al. (2009)[32]	RS	14 CCHS 31 controls	15.1 ± 2.3 15.1 ± 2.4	CCHS	Significant reduced volume in CCHS compared to controls	1 mm

Cabrera-Mino et al. (2020)[33]	RS	25 SVHD 38 controls	15.9 ± 1.2 16.0 ± 1.1	SVHD	Significant volume reduction in SVHD compared to controls (100%) *Significant lower MoCA and WRAML2 scores in SVHD over controls*	0.9 mm (resampled to voxel size 0.2 x 0.2 x 0.2 mm for volume)
Singh et al. (2019)[34]	RS	27 SVHD 35 controls	15.7 ± 1.2 15.9 ± 1.2	SVHD	Tissue injury (increased AD, RD and MD values) in SVHD compared to controls	0.9 mm
Muller et al. (2011)[35]	PS	120 patients	1.2 - 18	Masses	Surgical treatment of craniopharyngioma affects rather radical increase in BMI during the post-surgical 36-month period, especially hypothalamic involvement/lesion of the anterior and posterior hypothalamic area, i.e. involving the MBs and the area beyond MBs, with consequential severely lower QoL scores for social function family	Unknown
Freeman et al. (2004)[36]	RS	72 patients	22 months – 31 years	Masses	Displacement or distortion of MB was unilateral in 68% and bilateral in 19%. In 10 patients MB could be identified only unilaterally *The intimate relationship to the MB, fornix, and mammillothalamic tract suggests a role for these structures in epileptogenesis associated with hypothalamic hamartomas.*	2.5 mm; intersection gap 0.1 mm
Vanslambrouck et al. (2000)[37]	CR	1 patient	5	Masses	Dolicho-ectasia of the left posterior cerebral artery and internal carotid artery with compression of the brainstem, MBs and the optic tract.	Unknown
Roth et al. (2015)[38]	RS	45 patients (22 with HO 23 without HO)	Mean 13.9	Masses	Subjects who developed hypothalamic obesity (HO showed more frequently lesions affecting the third ventricular floor, MBs, and anterior, medial (all <0.05), and most importantly posterior hypothalamus (P < 0.01).	Unknown
Perez et al. (2021)[39]	RCT	35 patients: - 20 ExQW - 15 placebo	 16.9 ± 4,7 16.9 ± 4,3	Masses	MB intact in only 22% of the patients Greater MB injury associated with greater reductions in adiposity following GLP-1RA treatment	1 mm

125 Ozyurt et al. (2014)[40]	PS	15 patients 24 controls	Median: 17.3 (6) 17.6 (4.8)	Masses	4 / 5 patients with delayed-recall performance in the clinical range had postoperative hypothalamic lesions involving MBs owing to afferent and efferent projections, injury of the hypothalamus, including MBs, might lead to not only medial temporal and subcortical dysfunction, but also to frontal dysfunction, through diaschisis	Unknown
122 Mortini et al. (2016)[41]	RS	47 patients: - 10 children - 37 adults	34 ± 2 11 ± 1 41 ± 2	Masses	Excluding age, no significant differences between childhood and adult cases were observed for any of the variables. Hypothalamic syndrome was associated with the degree of MB involvement	Unknown
130 Goncalves et al. (2014)[42]	CR	1 patient	9	Encephalitis	increased T2 and FLAIR signal in the MBs	Unknown
91 Poloni et al. (2009)[43]	CR	1 patient	3 months	Metabolic disease	Contrast enhancement of the MBs	Unknown
93 Shah et al. (2020)[44]	CR	2 patients	26 months 21 months	Metabolic disease	bilateral symmetric optic neuritis and T2 hyperintensity of bilateral MBs, dorsal aspect of medulla and the area postrema	Unknown
90 Friederich (2020)[45]	CR	2 patients	4 & 8	Metabolic disease	Diffusion restriction in the posterior part of the hippocampi medial left temporal lobe, posterior inferior thalamus and the MBs in Leigh disease	Unknown
92 Inui et al. (2000)[46]	CR	1 patient	13	Metabolic disease	Low intensity of the MB on T2	Unknown
95 Oikawa et al. (2001)[47]	RS	15 patients	1-28	Epilepsy	Atrophy and/or signal change of the MB in 3 (20%) all accompanied by hippocampal and parahippocampal atrophy. *Patients with abnormalities of the circuit of Papez did not have more severe epilepsy than those without*	1.3 mm

Tschampa et al. (2011)[48]	RS	43 patients	2 – 79	Epilepsy	20 patients showed hyperintense pulvinar lesions; 4/20 showed atrophy of the MB and fornix (3 ipsilateral, 1 contralateral) 5 patients showed linear defects in the anterior thalamus; there was ipsilateral atrophy of the MB and fornix in all	Unknown
Kodama et al. (2003)[49]	RS	34 patients	3 - 54 (mean 28.6)	Epilepsy	Atrophy of the fornix (14.7%), MB's (17.6%), mamillothalamic tract (8.8%), and thalamus ipsilateral to the epileptic focus (11.8%) in patients with temporal lobe epilepsy	Unknown
Urbach et al. (2005)[50]	PS	45 patients 15 controls	3 - 66 18 - 54	Epilepsy	MB volumes were significantly smaller on the operated-on than on the non– operated-on side and significantly smaller in patients compared with controls. No volume differences of the MBs existed between seizure-free and non–seizure-free patients	1.1 mm
Ferreira et al. (2003)[51]	RS	115 patients	3.5 - 80	Epilepsy	MB volume loss in 5 (11.6%)	Unknown
Mamourian et al. (1993)[52]	CR	3 cases: 1 pediatric	4.5	Epilepsy	There was no parenchymal abnormality seen within the temporal or parietal lobes. However, the scan showed absence of the right MB	5 mm
Ng et al. (1997 [53]	PS	27 patients 10 controls	6 - 51 23 - 67	Epilepsy	In 19 cases there was unilateral abnormality in the hippocampus (HC); there was a smaller MB on the same side as the abnormal HC in all 19 cases. *Smaller fornix in 18 / 19 cases*	2 mm
Ozturk et al. (2008)[54]	RS	178 patients 353 controls	36 (8-69) 49 (7-87)	Epilepsy	Asymmetrical MBs in group with seizures (14%) and without seizures (6.5%)	4-5 mm
Hakyemez et al. (2006)[55]	PS	32 patients 42 controls	10 – 67 13 – 62	Epilepsy	There was a significant difference in volume of fornix and MBs of the patients versus control subjects ($p < 0.005$) MBs were abnormal in 37% of patients with bilateral involvement in 15%.	3 mm; gap 1 mm

Kim et al. (1995)[56]	RS	40 patients 34 controls	13-57 14-56	Epilepsy	Asymmetrically small MB was found in 3% (1 of 33) of the presurgical hippocampal sclerosis group and in 57% (4 of 7) of the postsurgical hippocampal sclerosis group, all 4 accompanied by asymmetrically small fornix on the same side In the control group none of the subjects had asymmetrically small MBs	2-3 mm
Kuzniecky et al. (1999)[57]	PS	50 temporal lobe epilepsy 10 extra-temporal lobe epilepsy 17 controls	17 – 42 Mean 30 24 - 41	Epilepsy	MB volumetric measurements indicated that 34 (41%) patients with MTLE (Group 1) had evidence of atrophy. Conversely, no patient with extratemporal lobe epilepsy had atrophy of these structures.	1.5 mm; no gaps
Grewal et al. (2018)[58]	RS	20 patients	17-66	Epilepsy	Mammillary body volume decline after LiTT is associated with better seizure outcomes (average volume reduction of 34.6% in the ipsilateral mammillary body after successful (seizure freedom at 1 year) amygdalo-hippocampal LiTT versus an average decline of only 8.4% in patients with poor outcomes after LiTT.	1 mm
Ciesielski et al. (1999)[59]	PS	10 patients 10 controls	6.8 – 13.5 6 - 13	Iatrogenic	Significant reductions in volumetric measures for bilateral MBs in patients compared to controls, significant reductions in prefrontal cortical volume, visual and verbal single-trial memory deficits, and visuospatial, but not verbal, multitrial learning deficits.	1 mm; no gap
Kwan et al. (2015)[60]	CR	1 patient	16	Iatrogenic	Clinical and imaging findings suggest that extra-ventricular Ommaya catheter position may lead to a direct methotrexate-induced toxicity to the Papez circuit. intense homogeneous enhancement of the MBs and corresponding hyperintensity in the MBs on T2-weighted images.	Unknown

Anand et al. (2015)[61]	CR	1 patient	28 months	Miscellaneous	Susceptibility within the MBs on T2 gradient echo imaging consistent with hemorrhagic necrosis in a patient with recurrent familial acute necrotizing encephalopathy (ANE1).	Unknown
Herebian et al. (2017)[62]	CR	1 patient	6	Miscellaneous	Global brain atrophy, hypointensities of globus pallidus, MBs, and cerebral peduncles, comparable to findings in neurodegeneration with brain iron accumulation disorders.	Unknown
Assis et al. (2018)[63]	CR	1 patient	'adolescent'	Miscellaneous	Small focus of encephalomalacia in the anteromedial aspect of the right thalamus, presumed chronic infarct, atrophy of the right MB and asymmetric volume loss of the retrocommissural fibers and anterior pillar of the right fornix. A small gliotic tract could be seen along the expected course of right mammillothalamic tract on a sagittal T1 reformatted image in a patient with complex partial seizures	Unknown

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